VOLUME 8 NUMBER 2

AMERICAN JOURNAL

OF

DISEASES OF CHILDREN

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AUGUST, 1914

PUBLISHERS

AMERICAN MEDICAL ASSOCIATION

CHICAGO, ILL.

Entered as Second-Class Matter, January 4, 1911, at the Post-Office at Chicago, Illinois, Under Act of Congress of March 3, 1879

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American Journal of Diseases of Children

Vol. 8 AUGUST, 1914 No. 2

PRIMARY PULMONARY ACTINOMYCOSIS IN A CHILD AGED TEN

WITH POST-MORTEM EXAMINATION *

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NEW YORK

Actinomycosis pulmonis as a primary condition, though uncommon in America, has been observed rather frequently abroad. In the German literature Aschoff, Heuser, Rutinmeyer, Köster, and Kashiwanuva report such cases in detail, the latter describing four instances, while Lindt described a single case of primary involvement of the apices. In America, case reports of only a very limited number of this type have been recorded, owing perhaps, to a great extent, to the relative infrequency of the various types of this ailment. The disease may be acute or chronic, local or general. The symptoms vary within wide limits; they may be pronounced or, on the other hand, indefinite.

Actinomycosis, as we know, is an infectious disease caused by a specific micro-organism, *Actinomyces bovis,* originally named "ray fungus" by Bollinger and Hartz. It must not be confounded with the so-called pseudotuberculosis, streptothrix or cladothrix infections or atypical actinomycotic processes described by J. H. Wright under the name of "nocardioses."

It was first recognized in man by Langenbeck in 1845, who described a case of vertebral caries—the pus containing numerous "sulphur-like" granules. Twelve years later, in 1857, a patient with thoracic involvement was reported by Lebert. Murphy of Chicago, in 1883, was the first clinician on this side to report a patient suffering with this disease. The identity of the human and bovine disease was first pointed out by Ponfick. In a masterly monograph, Israel, in 1885, proved that the disease was transmissible from man to animal. The experiments proved that the infection could be transmitted from man to rabbits. These observations were later confirmed by Ponfick, Wolff, Hanan, John and Boström. Hodenpyl (1890) deserves the credit of having reported the first case of pulmonary actinomycosis in America.

* Submitted for publication June 8, 1914.

The organism is widely distributed in nature and is easily isolated from certain grasses. Direct contagion is denied by many, though a few cases have been reported in which men have contracted it from animals. At the Royal Victoria Hospital one of the earliest cases occurred in the nurse attending a case of actinomycosis.

The infection may take place as follows: through (*a*) the mouth and pharynx, (*b*) the respiratory tract, (*c*) the gastro-intestinal tract, (*d*) the skin and wounds, and (*e*) idiopathically (?).

Pathologically, two types of lesions are recognized as characteristic of this disease, that is, (1) neoplastic and (2) inflammatory. Each type has selective tendencies in the special species chosen. Horses and cattle suffer more frequently from the former and man and hogs from the latter. The neoplastic type usually occurs in very vascular tissues and clinically resembles sarcomatous invasions, which may, however, result in spontaneous recovery. The inflammatory type which is most frequently seen in man, usually avoids parenchyma, spreads rapidly and forms characteristic sinuses from which the sulphur-like granules are discharged and can be shown microscopically to contain the "rays." Microscopically the actinomycotic area lends itself to division into three zones; from within outward they are: (*a*) the central zone of fungi and detritus, (*b*) the middle zone of round and epithelial cells which supplies the inner with detritus, and (*c*) the outer zone of granulation tissue.

The bacteriology is of especial interest to the laboratory worker, and although it is deserving of more than a passing remark, space does not permit any extensive references.[1]

The severity, location and type of the infection determines the nature of the symptoms. The sites are the following in their order of frequency: (1) cervical-facial, 55 per cent., (2) thoracic, 20 per cent., (3) abdominal, 20 per cent., of which more than half (50 to 60 per cent.) involve the cecum, and (4) cutaneous, 5 per cent. In the thoracic form definite signs and symptoms are usually absent until the disease is far advanced. Cough, with fetid sputum, consolidation and cavity formation may appear late, with attending cachexia, anemia and temperature of a variable type. The presence of an inflammatory swelling or a board-like induration of the thoracic wall is suggestive; later on suppurating foci or sinuses may result and arouse our suspicions as to the real nature of the process, which may be confirmed by a microscopic examination of the pus. Pulmonary cases, it has been asserted, are usually secondary to the abdominal variety. Because

1. For a full description of the micro-organisms of actinomycosis, the reader is referred to Wright, J. H.: Biology of the Micro-organisms of Actinomycosis, Jour. Med. Research, new series, 1905, viii, 349.

of the involvement of the right lower lobe in the majority of the cases it is thought that the lymphatic channels are to a great extent responsible for the transmission of the infection from the right side of the abdomen to this region. Infection through the blood-stream, however, seems more logical together with "locus resistentiae minoris" as in other infections, particularly so when the lung has been exposed to trauma.

The exact diagnosis can be readily established in many instances by microscopic recognition of the fungi from the sulphur-like bodies found in most cases. It is necessary that these granules be examined in thin preparations to avoid error—the liability "to look but to see not." Translucent bodies are positive evidences of such involvement, especially if club-shaped.

The prognosis, according to McKenty, varies with the extent, severity, and location—the nearer the surface or within the scope of radical surgical interference, the greater the possibility of early and successful cure. Therefore, it is evident, as this writer states, that cervical infections give a more favorable prognosis and are fatal in only 11 per cent. of cases, while on the other hand the abdominal mortality ranges as high as 71 per cent. and the thoracic even higher—83 per cent.

Treatment of actinomycosis is symptomatic and consists in the administration of potassium iodid continued over a long period of time. The drug appears, however, to act less favorably in the pulmonary type. The surgical treatment of this variety has not given brilliant results. The Roentgen ray is of doubtful value. Vaccine treatment has given some success after a considerable period.

CASE-REPORT

History.—J. B., a schoolboy, aged 10, of Italian parentage, was admitted to the Beth Israel Hospital (Dr. Huber's service), June 18, 1913, in a drowsy and restless condition, speaking only at times, always incoherently. The following history was obtained with difficulty through an interpreter. It was not until some time later that we learned that the boy was in the habit of spending much of his time in a stable.

Present Illness.—About four weeks before admission the child was struck on the chest by a man, with sufficient force to give rise to considerable soreness of the right side and pain, causing him to complain for a few days. Two weeks later he was suddenly seized with high fever, headache, anorexia, prostration, and vomiting, the latter continuing for three days after the onset. On the fourth day of the illness an unproductive cough developed and was soon followed by a thin brownish expectoration which afterward became greenish and fetid, and was later accompanied by dyspnea. Three days before admission he began to show evidences of restlessness, drowsiness and incoherence, which have been increasing.

Previous History.—The previous history of the patient may be summarized in the following few remarks. "Instruments" were necessary at his birth. He was breast-fed until one year of age. His habits were always good. He was never sick before. The family history is without importance.

Physical Examination.—The patient appeared fairly well developed and nourished. Marked dyspnea, great prostration and extreme facial pallor were observed immediately on approaching his bed, together with a disagreeable fetid odor which pervaded that section of the ward. The skin was warm and dry. Eye examinations were negative. Extensive herpes labialis was present. The tongue was thick and coated, brownish white, and the pharnyx was congested. The teeth appeared normal.

Thorax: Since, in the history, the chief complaints centered in the respiratory apparatus, the thorax and its contents received particular attention. The movements of the right chest-wall were greatly diminished during both acts of respiration. There was marked induration of the anterior and lateral aspects of the right side of the chest, particularly below. Though the regions involved felt hard, there was no evidence of local inflammation, no redness, no tenderness, increased local heat or deep-seated fluctuation. The interspaces were more prominent on the opposite side. Percussion over the affected side gave a

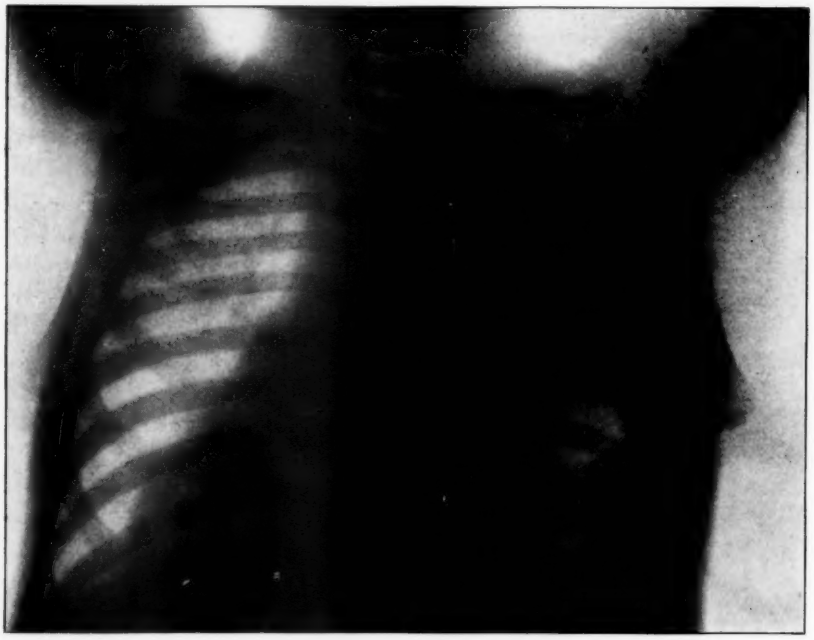


Fig. 1.—Pulmonary actinomycosis, showing extensive involvement of right side.

"boardy" note which was diminished from above downward. The breath-sounds were very distant and bronchial in character, heard best after coughing. The voice and whisper sounds were also of this type. Fine and large moist râles accompanied the breath-sounds. These physical signs extended to the lower lobes but also diminished in intensity. Over the whole left lung there were areas of compensatory exaggeration of the normal findings together with scattered portions of bronchial breathing and fine moist râles.

Examination of the heart showed it to be in the normal position.

Abdomen: The right lobe of the liver was pushed down about an inch below the costal margin; otherwise the findings here were normal.

Course.—On admission to the hospital, the patient's temperature was 103.6 F. It continued remittent, ranging from 101 to 105, with a corresponding increase in the pulse-rate. The respirations varied from thirty to fifty per minute.

The blood examination showed 12,000 leukocytes with 84 per cent. polynuclears and 16 per cent. mononuclears (150 cells counted). Four days later a second blood count gave 9,200 leukocytes with 79 per cent. polynuclears and 21 per cent. mononuclears (the second count was made after the thoracotomy). Therefore, during a period of four days, the patient's infection was becoming exceedingly severe and his resistance poorer.

The urinalyses were negative.

A roentgenogram disclosed a dense white shadow occupying the upper three-fourths of the right pulmonary field, which was interpreted as fluid—perhaps a lung abscess or a sacculated empyema.

Many specimens of sputum were examined microscopically by the members of the medical staff, but no "ray fungi" were found. (Lebedeff, Machinskit, and Sokoloff mention cases which were diagnosed from the sputum findings.) The reports in each instance in our case showed polynuclear leukocytes, elastic tissue, streptococci, and staphylococci. Macroscopically the sputum was greenish yellow, thick, semisolid, tenacious, and foul-smelling. The odor was characteristic of gangrene of the lung and accordingly the case was regarded as extensive pulmonary gangrene, the cause of which was not evident.

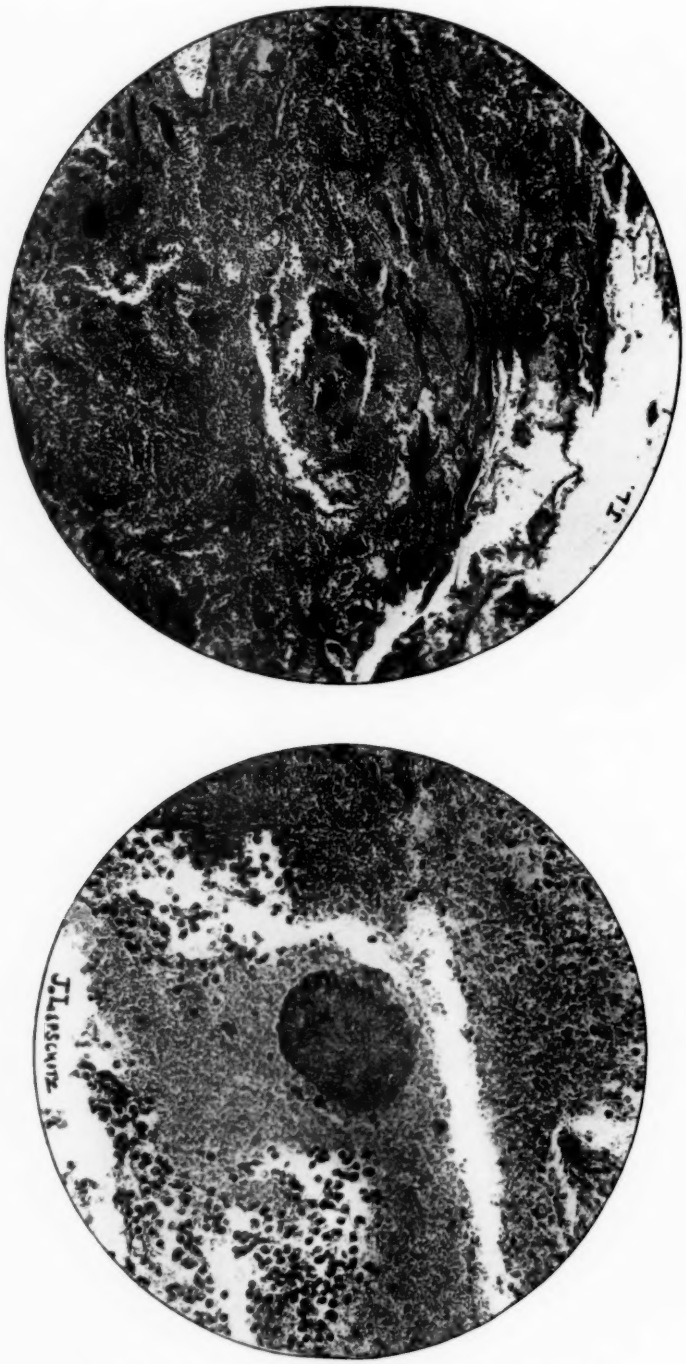
Operative Procedures.—In view of the physical signs, clinical course, and roentgenographic findings, the chest was aspirated in the sixth right interspace in the midaxillary line. After several attempts with needles of increasing caliber, 8 c.c. of creamy greenish yellow, tenacious and very foul-smelling pus were evacuated. This was later reported on fresh smear examination to contain staphylococcic and Gram-positive diplococci. Culturally there was no growth but some anaerobic bacteria were present.

The thoracotomy, preceded by an exploratory aspiration, the needle being left *in situ* as a guide, was performed the same evening under nitrous oxid and oxygen anesthesia. The site chosen was in the same area from which the pus had been previously obtained. A pint of thick creamy pus was slowly evacuated. The work had to be done rapidly as the condition of the patient even while on the operating-table, was precarious. A large rubber tube was quickly inserted, the dressing applied and the child taken to the ward.

His condition continued serious with attacks of restlessness, delirium and stupor lasting a week, the patient dying from toxemic paralyses and exhaustion.

The history of trauma to the chest and the obscure etiology of the pulmonary gangrene in an apparently well-developed boy aroused great interest; there was also a strong suspicion that external violence had been a contributory factor. The condition was therefore reported to the coroner's office in the hope that we might perhaps get an opportunity through necropsy to ascertain, if possible, the cause of this illness. A post-mortem examination was performed by Dr. Otto Schultz, who, after having seen gross sections of the gangrenous right lung, suggested the possibility of actinomycosis; but not until a thorough search for other foci was made (an important point in the opinion of McKenty), did he consider the disease as of primary origin in the lungs. The diagnosis was later corroborated by stained microscopic sections. A detailed gross pathologic report of the condition in the lungs follows:

Necropsy Findings.—The right upper lobe is very large and completely solid. Its pleura is thickened and covered everywhere with fresh fibrin. On cross-section this lobe is almost completely gangrenous, green, and honeycombed with abscesses containing yellowish-green pus, the largest of them being the size of a hen's egg. These abscesses are not encapsulated, but are lined by dirty irregular shreds of lung tissue. The lower lobe is studded with numerous patches of bronchopneumonia and from the center of the majority of these patches small amounts of pus can on pressure be forced out. The remainder of the lung is solid with reddish-gray hepatization. The left lung is covered with a thin layer of fibrin on its outer surface. This, on section, shows numerous patches of red



Figs. 2 and 3.—Actinomycosis pulmonis (primary).

hepatization which are wide-spread. The bronchial lymph-nodes are larger and softer than normal.

The microscopic examination by Dr. Eli Moschcowitz, pathologist to the Beth Israel Hospital, gives the following condensed report:

The pulmonary alveoli are completely filled with exudate, in part consisting of almost pure fibrin and in part with fibrin and large round mononuclear cells. Scattered throughout the consolidated lung are discrete darker areas which under low power resemble miliary abscesses. These areas vary in size from the head of a pin to a millet-seed and consist of collections of small round cells in the center of which are "ray fungi" varying in number from one to a dozen. The lung tissue in these areas is absent, the wall consisting of partially destroyed consolidated alveoli. The exudate immediately surrounding the "ray fungi" is completely necrotic. The walls of the lung alveoli are normal. The pleura is covered by a thin layer of hyalinized fibrin.

In reviewing rather hastily the more recent literature, we find the disease to be rare in children. In McKenty's series of thirty-seven cases seen in America (nine in females) occurring within 100 miles of Montreal, the youngest patient was a boy, aged 10. The incubation period varies from a few days in the cases occurring in the jaw to weeks and months in the abdominal and pulmonary varieties. It has been found, as a result of observation and experiments, that infection does not occur unless there is a lesion in the skin or mucous membrane.

Foulerton lays stress on the fact that, in many cases, an exact diagnosis on clinical evidence is impossible until the chest or abdominal walls are involved by extension of the process outward. Perforation of the skin then occurs and the organism may be discovered in the secretions on microscopic examination.

Incidentally we may refer to the disappointment in not being able to find or isolate the organism in the sputum or other secretions in some instances. The difficulties in the positive identification of the parasite by laboratory methods may be due to several causes: in the first place there is extreme difficulty of obtaining growth of many parasites of this class on artificial mediums, and, secondly, because of the varying morphology at different phases of the life-cycle of the parasite, recognition is not easy. It is well known that the mycelial or "ray-fungus" form represents only one of the three forms in which the parasite occurs. The typical mycelium and the mycelium in the earlier stages of "fragmentation" when it is breaking up into "rod forms," represent the only stage at which the parasite can be recognized positively by the microscope. At a later stage when sporulation has occurred, the irregular "bacillary" form and spherical spores resembling the pus cocci are seen, both staining deeply by the Gram method.

281 East Broadway.

CASEIN IN INFANT-FEEDING

THE PREPARATION OF DRY POWDERED PARACASEIN AND PRELIMINARY EXPERIMENTS CONCERNING ITS USE AS THE PRINCIPAL PROTEIN CONSTITUENT IN INFANT FOOD *

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WITH THE ASSISTANCE OF

B. H. RAGLE, A.B.

In a previous communication[1] we mentioned the preparation of dry powdered paracasein and showed that it was completely digested when fed as a constituent of infant food. It seems desirable to give the details of the method used in preparing this substance, together with additional experiments involving its use.

I. METHOD OF PREPARING DRY POWDERED PARACASEIN

Fat-free milk is curdled by the addition of rennin and when the curd has become so firm that it will make a clean break when the finger is thrust diagonally into it and then lifted up, divide it into small pieces, remove the whey and wash several times with water. If the curd is obtained from a dairy, or for any reason becomes matted together, it must be broken up and then passed through a meat chopper.

The finely divided curd is now placed in a large vessel such as a cheese vat or a clean wash tub, and to it is added five volumes of water for every volume of milk used to produce the curd. The curd is now dissolved by adding about 10 c.c. of concentrated ammonia water for each quart of milk used. As the curd goes into solution, rather slowly, more or less continuous agitation will be necessary.

After complete solution the paracasein is precipitated in the following manner: Take 2.5 c.c. of glacial acetic acid for every quart of milk used and dilute it with 25 volumes of water. Add this diluted acid to the solution of paracasein, a few cubic centimeters at a time,

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* Submitted for publication, April 22, 1914.

* The expense of this investigation was defrayed by the Boston Floating Hospital, Boston, Mass. The chemical work was done in the Biochemical Laboratory of the Harvard Medical School, Boston, Mass. We wish to thank Dr. Otto Folin for his courtesy in extending this privilege to us.

1. Bowditch, H. I., and Bosworth, A. W.: AM. JOUR. DIS. CHILD., 1913, vi, 394.

and with constant agitation. This addition of acid will precipitate the paracasein and very close attention is required at this point, for an excess of acid will redissolve the precipitated paracasein which should separate out as a large flocculent precipitate quickly settling to the bottom of the vessel. If a very fine precipitate is formed, which does not settle rapidly, it is an insoluble calcium salt of paracasein[2] and the addition of a little more acid will change it to the desired form, free paracasein. More or less acid than the amount specified may be required to give the proper precipitate, depending on the condition and age of both the milk and the curd and on the amount of ammonia used to dissolve the curd. After the paracasein has settled and the supernatant liquid has been removed, it is washed several times with water and then redissolved, using the same volume of water as before, but using caution at this point so that an excess of ammonia is not added, for this would cause hydrolysis with consequent loss in yield of paracasein. It is best to dilute the ammonia water with fifty volumes of water and use just enough of this diluted ammonia to dissolve the paracasein. After complete solution the paracasein is again precipitated with dilute acetic acid and washed as before.

The procedure from this point on depends on how pure a product is desired. At each precipitation some insoluble calcium paracaseinate is formed and carried down with the large mass of free paracasein and it is only by repeated precipitations that the calcium is all removed. In order to secure practically ash-free paracasein it will be necessary to make six or seven precipitations.[2] Two precipitations, if properly made, will give a fairly good product.

After the final precipitation and washing the paracasein is placed in a linen bag and allowed to drain for two hours. It is then transferred to a mortar and triturated with 95 per cent. alcohol. After allowing to settle, decant off the alcohol and again triturate with a fresh portion of alcohol, repeating two or three times. The dehydration is completed by allowing the paracasein to remain in contact with a large volume of 95 per cent. alcohol for an hour or two. This alcohol is removed by decantation and filtering through the linen bag and if desired the bag and its contents may be placed in a press to remove most of the remaining alcohol. The preparation is now made to pass through a 20-mesh sieve and finally dried at a temperature between 80 and 90 C.

With proper care as to technic the final product will be a fine powder. If a powder is not obtained it can be secured by grinding in a coffee mill. If grinding is necessary the material should be placed in

2. Van Slyke and Bosworth: Jour. Biol. Chem., 1913, xiv, 203.

the drying oven again in order to remove any alcohol which might be held inside the larger particles.

One product made by us with two precipitations contained 1.7 per cent. moisture, 0.4 per cent. ash and 98 per cent. paracasein. It was insoluble in water, slightly soluble in a 5 per cent. solution of sodium chlorid and completely and quickly dissolved by a dilute solution of sodium carbonate. The substance prepared in this manner is paracasein, not casein. The only difference between paracasein and casein is in the size of the molecule. One molecule of casein is split, by rennin, into two molecules of paracasein and no other substance is produced as was formerly believed.[3] This is quite similar to the production of two molecules of dextrose from one molecule of maltose.

II. THE USE OF DRY POWDERED PARACASEIN AS THE PRINCIPAL PROTEIN CONSTITUENT OF INFANT FOOD

1. *Low protein feeding.*—As we wished to secure additional data on the digestibility of dry powdered paracasein and also learn to what extent it might be used to supply the protein in infant food, we secured the data given below. The child selected for this experiment was 8 months old, weighed 4,480 grams and was in good physical condition. He had been receiving 1,470 c.c. each twenty-four hours, of the following mixture:

Sixteen per cent. cream	160 c.c.
Buttermilk	800 c.c.
Sterile water	640 c.c.
Milk-sugar	53 gm.

A study of his nitrogen metabolism was made during four days while he was receiving this food and the figures given in Table 1 under the heading "Buttermilk Period," are averages representing the average figures for a twenty-four-hour period. The food was then changed to one made according to the following formula:

Sixteen per cent. cream	161 c.c.
Distilled water	1,439 c.c.
Lactose	89 gm.
Paracasein	25 gm.

It will be noticed that the mineral constituents in the last formula are supplied by the whey carried in the 161 c.c. of 16 per cent. cream. We will call attention to this point in another paper, simply stating here that the low sodium chlorid excretion while the child was on this food was due to the small mineral intake.

The protein in the above formula is not all paracasein for 161 c.c. of 16 per cent. cream will contain about 4 gm. of protein of which 2.8 gm. will be casein and 1.2 gm. albumin. The 2.8 gm. of casein

3. Bosworth: Jour. Biol. Chem., 1913, xv, 231.

is equivalent to an equal amount of paracasein, so that out of the 29 gm. of protein in the formula all but 1.2 gm. can be considered paracasein. Just what favorable influence this 1.2 gm. of albumin might have on the utilization of the paracasein we are unable to state at this time.

The child took the food well after the first few feedings and seemed to maintain his general good condition during the nine days he received it. The nitrogen metabolism while on this food is represented by the figures in Table 1 under the heading "Paracasein Period." These are average figures for a twenty-four-hour period.

TABLE 1.—Low Protein Feeding. Urine Analysis; Average Figures for Twenty-Four-Hour Periods

	Buttermilk Period	Paracasein Period
Volume in c.c.	832.0	1110.0
Total acidity as N/10	128.4	46.9
P_2O_5, gm.	1.002	0.395
Chlorids as NaCl, gm.	1.100	0.165
Total nitrogen, gm.	2.020	1.335
N as NH_3, gm.	0.311	0.205
N as urea, gm.	1.544	0.782
Uric acid, gm.	0.073	0.056
Creatinin, gm.	0.020	0.020
Creatin, gm.	0.028	0.025
Nitrogen in feces, gm.	0.365	0.173
Gain in weight per twenty-four hours	41.2	13.3

A study of Table 1 indicates no marked change in the general nitrogen metabolism with the change of food and no toxic effect was observed. It will be noticed that the amount of nitrogen in the feces during the paracasein period is less than half that found during the buttermilk period, indicating better digestion and absorption of the paracasein. The average daily increase in body weight during the paracasein period was less than during the buttermilk period. The low phosphorus and chlorin eliminations during this period would indicate that this was due to a lack of sufficient inorganic material in the food. We hope to publish work on this before long.

2. *High protein feeding.*—It occurred to us that the dry, powdered paracasein might be combined with whey, or milk, and cream to make preparations quite similar to Finkelstein's formula. We made several experiments along this line and will report two of them here because they seem to possess special significance. One child was fed a high protein food containing plenty of whey and the other child a

high protein food containing an extremely small amount of whey. The two formulae are as follows, each being a twenty-four-hour quantity:

Formula 1.—Fat, 1.5 per cent.; sugar, 6 per cent.; protein $\frac{\text{.25 whey}}{\text{6.5 dry casein}}$

Twenty per cent. lactose solution	337 c.c.
Thirty-two per cent. cream	44 c.c.
Whey	355 c.c.
Water	662 c.c.
Lime-water	21 c.c.
Paracasein	93.0 gm.

Formula 2.—Fat, 1 per cent.; sugar, 6 per cent.; protein $\frac{\text{0.4 whey}}{\text{6.5 dry casein}}$

Twenty per cent. lactose solution	384 c.c.
Thirty-two per cent. cream	68 c.c.
Water	967 c.c.
Paracasein	96.0 gm.

In each case a study of the nitrogen metabolism was made during a preliminary period on a "normal" diet and also during the period of paracasein feeding. In no case was all the paracasein indicated in the formula ingested during a twenty-four-hour period, for it was quite impossible to manipulate the feeding-bottle so that the infants received all the paracasein; more or less always remained adhering to the walls of the bottle. The figures for total nitrogen in the urine, however, indicate the ingestion of large amounts of protein. The urines were collected in twenty-four-hour quantities and the analytical data obtained are given in Tables 2 and 3.

CLINICAL REPORT (RÉSUMÉ)

Experiment 1 (*High Protein and Whey*).—A male child 7 months old weighing 11 pounds 1 ounce. Diagnosis, rachitis, otitis media chronica. His digestion was normal and he was gaining weight. With the administration of special formula no untoward effect was seen until the last day or so, when diarrhea set in. The child's disposition and general condition remained good. He took his food fairly well. Although calorically feeding 120 calories per kilo, he lost during the experiment 9 ounces. (Unfortunately he died ten days later of bronchopneumonia.)

Experiment 2 (*High Protein and Very Small Amount of Whey*).—A male child 7 months old weighing 13 pounds, 13½ ounces. Diagnosis, normal child and feeding; well developed and nourished. This patient took his food well at all times, showed but little regurgitation and no indigestion nor diarrhea. There was no fever at any time. Although getting 120 calories per kilo he lost 1 pound in seven days during the experiment. (This patient was discharged well.)

The high protein feeding resulted in a greatly increased elimination of ammonia in both cases. In Table 3 the creatin figures are fairly constant, while in Table 2 quite a marked change is noticed in the creatin excretion with the beginning of the high protein period. We are unable to suggest any explanation for this.

TABLE 2.—High Protein Feeding with Abundance of Whey

URINE ANALYSIS

Twenty-Four-Hour Period Ending	Body Weight, lbs.	Volume, c.c.	Specific Gravity	Total Acidity as N/10	Chlorids as NaCl, gm.	Total N, gm.	N as NH_3, gm.	Uric Acid, gm.	Creatinin, gm.	Creatin, gm.
August 3 ...	.....	540	1.007	*	0.434	2.147	*	0.0864	0.0210	0.0454
August 4 ...	.....	500	1.005	48.0	0.684	1.624	0.328	0.0603	0.0216	0.0313
August 10 ...	11 1/16	270	1.013	*	0.210	1.663	*	0.0951	0.0150	0.0095
August 11 ...	.....	510	1.015	*	0.410	5.483	*	0.0704	0.0180	0.0069
August 12 ...	.....	620	1.015	38.4	0.149	5.034	0.521	0.0992	0.0190	0.0064
August 13 ...	.....	560	1.018	39.2	0.320	5.457	0.533	0.1066	0.0218	0.0049
August 14 ...	.....	430	1.015	*	0.189	5.058	*	0.0813	0.0164	0.0054
August 15 ...	.....	650	1.018	6.5	0.286	7.826	0.874	0.0819	0.0188	0.0100
August 16 ...	10 1/2	620	1.017	3.7	0.355	8.575	0.868	.....	0.0174	0.0061
August 17 ...	.....	600	1.017	....	.....	8.467	0.622	.....	0.0212	0.0072

* Alkaline.

TABLE 3.—High Protein Feeding with Small Amount of Whey

URINE ANALYSIS

Twenty-Four-Hour Period Ending	Body Weight, lbs.	Volume, c.c.	Specific Gravity	Total Acidity as N/10	Chlorids as NaCl, gm.	Total N, gm.	N as NH_3, gm.	Uric Acid, gm.	Creatinin, gm.	Creatin, gm.
August 1 ...	.....	420	1.008	52.9	0.341	1.705	0.219	0.0643	0.0200	0.0130
August 2 ...	.....	520	1.005	54.4	0.327	0.895	0.267	0.0688	0.0208	0.0169
August 10 ...	13 lbs., 13 1/2 oz.	950	1.008	79.8	0.794	6.118	0.798	0.0950	0.0241	0.0186
August 11 ...	.....	960	1.014	61.4	0.483	8.602	1.317	0.0658	0.0230	0.0121
August 12 ...	.....	800	1.016	89.6	0.378	8.823	0.560	0.1240	0.0240	0.0192
August 13 ...	.....	840	1.015	67.2	0.091	7.762	0.592	0.1091	0.0247	0.0220
August 14 ...	.....	730	1.018	29.2	0.000	9.403	1.226	0.0898	0.0234	0.0167
August 15 ...	.....	530	1.020	63.6	0.091	9.834	0.564	0.1219	0.0225	0.0151
August 16 ...	12 lbs., 13 1/2 oz.	630	1.017	44.1	0.025	7.444	0.494	.....	0.0209	0.0175

In Table 3 on August 14 it will be noticed that the urine was chlorin-free, chlorin appearing in the next twenty-four-hour specimen. This appearance of chlorin-free urine was not accompanied by symptoms of any kind.

The most surprising feature of these experiments in high protein feeding was the fact that no toxic or other unfavorable condition was manifested by the child receiving the small amount of whey. The extremely small amount of whey in the formula used for this experiment would lead one to expect the appearance of some such condition as that noticed by Holt, Levene, et al.,[4] when they fed their high protein "synthetic" food. The non-appearance, in our experiments, of the symptoms noted by them would suggest that some constituent other than the protein was responsible for the results obtained by them; possibly the 250 c.c. of tenth-normal sodium hydroxid (equal to 1 gm. NaOH) used to dissolve the curd, a constituent of their high protein "synthetic" food.

SUMMARY

1. A method is given for preparing dry powdered paracasein for use in infant-feeding.

2. It is shown that this paracasein is very easily digested and absorbed, and judging from the elimination of creatinin and creatin, it seems possible to maintain normal nitrogen metabolism by its use in infant-feeding.

3. It is shown that disturbances (fever, toxic symptoms, etc.) do not result from feeding this paracasein in exceptionally large quantities for seven days, even if fed with only the small amount of whey carried in 68 c.c. of 32 per cent. cream.

4. Holt, Levene et al.: Am. Jour. Dis. Child., 1912, iv, 265.

THE PRESENCE OF LACTIC ACID IN THE URINE IN CYCLIC VOMITING OF CHILDHOOD*

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Although the etiology of recurrent or periodic vomiting of children has been attributed to a variety of abnormal conditions none of the numerous theories advanced offers an adequate explanation for the cause of the symptoms observed.[1] The more recent ideas tend to correlate recurrent vomiting with some defects in metabolic processes, notably those concerned with carbohydrate transformations. Thus, in the pathologic state under discussion, diacetic acid and acetone in the urine are particularly prominent as abnormal constituents of that excretion. Inasmuch as carbohydrate deficiency in inanition in childhood leads rapidly to the same urinary findings, one may query with a great deal of pertinence whether these compounds mentioned can be regarded as factors in the etiology of cyclic vomiting, for they may indicate merely the effect of withdrawal of food, especially carbohydrates.

The purpose of the present communication is to report a case of cyclic vomiting showing one feature hitherto unrecognized, namely, the presence of lactic acid in the urine.

History.—The subject, a boy aged 2½ years, had been visited by one of us (Steele) at intervals since he was 3 months old. He had had several attacks of vomiting at irregular periods accompanied by a temperature ranging from 100 to 104 F. Up to the period of vomiting for which the urinary findings are presented below the child had been well fed, and attempts, which were partially successful, were made during the period of the attack to maintain a carbohydrate-rich diet. We therefore feel convinced that complete inanition did not play a significant rôle in the present instance. January 20 vomiting commenced, with a temperature of 104 F. at 10 a. m. The physical examination was negative. Calomel was given with good results. The temperature at night rose to 106 F. January 21 the bowels were loose and the temperature was 98.8 F. The child was apparently in normal condition on January 22. The twenty-four-hour specimens of urine were subjected to the analyses indicated in the accompanying table, the usual methods employed in this laboratory being followed.

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*From the Sheffield Laboratory of Physiological Chemistry, Yale University, New Haven, Conn.

*Submitted for publication, April 10, 1914.

1. Sedgwick: See AM. JOUR. DIS. CHILD., 1913, ii, 209, where the literature is reviewed.

Lactic acid was isolated as the zinc salt, according to the well-known method.[2] The salt was identified by a determination of the water of crystallization and the content of zinc oxid. From the twenty-four-hour specimen of January 20 more than 0.56 gm. zinc lactate was obtained.

Calculated for	$Zn(C_3H_5O_3)_2 + 2H_2O$ Per Cent.	Found, Per Cent.
H_2O	12.9	12.7
ZnO	33.33	33.43

At intervals between attacks of vomiting lactic acid was absent from the urine.

TABLE SHOWING URINE ANALYSIS DUR—

Date, Jan. 1913	Volume, c.c.	Specific Gravity	Reaction to Litmus	Total N., Gm.	NH_3 N., Gm.
20	600	1.020	Acid	6.30	0.13
21	600	1.020	Acid	5.54	0.14
22	620	1.015	Acid	5.20	0.20

In addition to lactic acid, diacetic acid, acetone and protein were present in the urine of January 20, all gradually disappearing as the attack passed off. An unusual feature in the present case is the presence in the urine of significant quantities of acid bodies—diacetic and lactic acids—without an appreciable rise in ammonia excretion. Ordinarily increased acid elimination is accompanied by augmented ammonia output, since ammonia is employed by the organism as a neutralizing agent. It is therefore evident that in our case some alkali other than ammonia must have been used for this purpose.

The significance of lactic acid in the urine is not well understood, although its presence in the urine in eclampsia,[3] pernicious vomiting of pregnancy,[4] phosphorus poisoning,[5] etc., is well known and its appearance may probably be taken as an indication of disturbed carbohydrate metabolism either induced by the pathological state itself or as a result of an accompanying inanition. Its meaning with respect to the etiology of recurrent vomiting of childhood must be left, therefore, for future investigation.

2. Hoppe-Seyler-Thierfelder: Handbuch der chemische Analyse, Ed. 7, p. 449.

3. Zweifel: Arch f. Gynäkol., 1905, lxxvi, 537.

4. Underhill: Jour. Biol. Chem., 1906-07, ii, 485.

5. Mandel and Lusk: See Am. Jour. Physiol., 1906, xvi, 129, for literature.

From the table it will be noted that creatin was present throughout the period of observation. In the intervals between attacks it was also a constant constituent of the urine, being present to the extent of 30 to 50 mg. creatin per 100 c.c. of urine. As the work of Rose, Folin[7] and Krause[8] has demonstrated, creatin is normally present in the urine of young children and it is therefore unnecessary to connect it in any way with the etiology of cyclic vomiting.[1] An increased elimination during an interval of attack with its usual accompanying inanition may be explained on the hypothesis of carbohydrate withdrawal.[9]

—ING AN ATTACK CYCLIC VOMITING

Creatinin, Mg.	Creatin, Mg.	Protein	Diacetic Acid and Acetone	Indican	Rotation Ventzke Degrees
210	102	Positive	Positive	Trace	—1.5
486	324	Trace	Trace	Trace	—0.6
176	177	Negative	Negative	Trace	0.0

6. Rose: Jour. Biol. Chem., 1911, x, 265.
7. Folin: Jour. Biol. Chem., 1912, xi, 253.
8. Krause: Quart. Jour. Physiol., 1913, viii, 87.
9. Graham and Poulton: Proc. Royal Soc., London (B) 1914, lxxxvii, 205.

These authors have recently criticized severely all estimations of creatin in urines containing diacetic acid.

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A CLINICAL STUDY OF TYPHOID FEVER IN CHILDREN *

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The fact that children have not been supposed to be very susceptible to typhoid fever, and, when affected, are apt to exhibit an irregular type of the disease, would seem to render a study of cases of value for future comparison and reference. It is during an epidemic that they are most liable to be attacked, so that such an occasion affords the most favorable opportunity for a study of any peculiarities the disease may show in early life. Since the employment of Widal's test, the field has widened by proving that typhoid fever in certain mild types may be overlooked in children or mistaken for some other affection.

During the fall of 1913, an epidemic of typhoid fever occurred in the lower East side of New York, including 521 reported cases, with a mortality of 11.7 per cent. Many children were attacked as shown by the following reported number of cases: Under five, 36; five to nine, 98; ten to fourteen, 87. These cases, I think, show a much larger proportion of attacks among children than have usually been reported. A little over two-fifths of all the cases in this epidemic occurred in children under 14 years of age. Dr. Osler in reporting on 1,500 cases of typhoid fever in Johns Hopkins Hospital states that 231 were under 15 years. Dr. Ogan of the New York Health Department, in a study of 1,500 cases in 1912, to compare with Osler's series, found 437 under 15 years. In a personal communication to the writer, Dr. Ogan states that children under 12 constitute one-quarter of the population of New York City and that approximately one-quarter of all cases of typhoid fever occur under 12 years, so that children contribute their fair ratio to this disease. It is evident that we must revise our ideas as to the frequency with which typhoid fever occurs in children.

The cases occurred during August, September, October and November and the Board of Health succeeded in tracing the outbreak to infected milk. It was found that 10 per cent. of the people living in the district were supplied by one milk company and that 70 per cent. of the cases reported were getting milk from one dairy. A case of typhoid fever was found in the vicinity of this dairy, although not

* Submitted for publication, June 8, 1914.
* Read before the American Pediatric Society at Stockbridge, Mass., May 26, 1914.

directly connected with it, and it is highly probable that the infection was carried by flies. When the milk from this source of supply was cut off by the Health Department, the epidemic ceased. The fact of it being a milk outbreak probably accounts for the large number of children attacked.

The eleven cases here reported were treated in the children's wards of the New York Postgraduate Hospital. The ages were as follows: 2 years, 1 case; 3 years, 2 cases; 4 years, 2 cases; 5 years, 2 cases; 6 years, 3 cases; 8 years, 1 case.

The duration of the fever, including the period before admission, as given in the histories, was varied and ran, from the shortest to the longest period, as follows: 7 days; 11 days; 16 days; 18 days; 19 days (two cases); 20 days; 21 days; 24 days; 33 days; 42 days. In only five cases did the temperature run very high during the active stage, two reaching 106 F., one 105.2 F. and two ranging from 104 F. to 105 F. The temperatures were not high in the other cases and were distinctly remittent toward the close of the disease. The diagnosis was confirmed in every instance by a positive Widal and no case showed any rose spots.

A study of the blood on admission is shown in the following table:

TABLE 1.—Results of Blood Examination on Admission

Age Years	Reds	Whites	Polymorphonuclears Per Cent.	Lymphocytes Per Cent.
8	5,200,000	7,200	58	42
5	4,028,000	5,700	65	35
3½	4,352,000	10,000	81	18
4	4,088,000	4,200	52	48
2	4,130,000	9,100	68	32
3	4,270,000	13,700	68	32
6	4,848,000	9,200	82	16
6	4,600,000	7,400	64	36
4	4,800,000	10,100	74	26
6	4,264,000	8,700	79	21
5	4,600,000	6,000	64	35

An inspection of this table shows the red corpuscles to be about normal in numbers. There is no appreciable diminution of the leukocytes except in one case, a child of 4 years, in whom they numbered only 4,200. These cases thus failed to show the leukopenia that is supposed to accompany typhoid fever. In the differential count, a study of the polynuclears shows what may be considered a polynucleosis when we have in mind the age of the patients. In every instance the percentage is appreciably higher than the average for the age. The two most marked were in a 3-year-old child showing 81

per cent. against a normal of 42 per cent., and a child of 6 years with 82 per cent. against a normal of 52 per cent.

The lymphocyte count was more irregular, being normal in only one case, a child of 6 years, with 36 per cent. In two cases, the count was above the normal for the age, one child of 8 years giving 42 per cent. against 33 per cent. average, and another of 4 years showing 48 per cent. against 41 per cent. average. The remaining eight were below the normal for the age. In two of the cases the relative diminution was very marked, one child of 3 years having only 18 per cent. and another of 6 years 16 per cent. against 47 per cent. and 37 per cent. averages for these ages.

A study of the urine showed that the kidneys were not much affected in this series; six cases were entirely negative, three gave traces of albumen, with occasional hyaline and granular casts, and a small number of pus cells were also reported in four of the cases.

The gastro-intestinal symptoms were neither marked nor severe. Vomiting was noted at the onset in four cases and during the fever in one case. Five of the patients had diarrhea, which was more occasional than constant; five were constipated most of the time, and one showed no abnormality. They seemed both to enjoy and digest the generous dietary that was given. For a number of years the opinion has been gaining ground that typhoid fever patients were being fed on a too exclusively weak and liquid diet. While the latter was based on the idea that a bland, fluid diet was safest for an inflamed, irritated bowel, the loss of weight and extreme inanition that sometimes followed this plan had unfortunate results. Some patients cannot take sufficient milk or other fluid nourishment to repair the waste caused by the fever and keep up the general vitality. It must be remembered that all digestible food is in liquid form when it passes from the duodenum into the ileum, and that feces, consisting largely of dead bacteria, epithelial cells and the secretions of the bowel, usually become solid only in the lower part of the colon. It is difficult to see how such material can have any appreciable effect on ulcers usually located in the ileocecal region. A more generous and varied diet not only stimulates the jaded appetite and digestion, thus making the patient able and willing to take more nourishment, but the resisting powers are thereby increased and the dangers of relapses thus lessened.

Acting on this idea, the children were allowed the general ward diet with the exception of meat. This included milk, cocoa, eggs, bread, toast, crackers, cereals, jelly, potatoes, gravy, broths, custard, junket, apple sauce, orange juice, ice-cream and lady fingers. They received nourishment once every three hours throughout the day.

An effort was made to give them forty calories per kilo body-weight per day, but in not a single instance was it possible to make

them take the full required number of calories through the febrile stage. In some instances, sugar of milk or malt-sugar was added to the liquids in order to increase the total caloric value of the food but this also failed, since the children refused to take it. Dr. Dennett, who had this feature of the work in charge, concluded that while from a theoretical standpoint it would be beneficial to have them take the required number of calories, it was not practicable to carry out the caloric feeding in these cases, since children, unlike adults, cannot be forced to take food.

The success of this varied diet is shown in Table 2.

TABLE 2.—DATA CONCERNING TYPHOID PATIENTS ON LIBERAL DIET

Age Years	Weight			
	On Admission Pounds	On Discharge Pounds	Gain Pounds	Loss Pounds
8	43 1/4	41 1/8	....	2 1/8
5	30 5/8	32 3/4	2 1/8	
3 1/2	31 1/8	33 7/8	2 3/4	
4	29	32 5/8	3 5/8	
2	24	23 3/4	....	1/4
3	22 3/8	30 3/8	8	
6	40 9/16	38 3/8	....	2 3/16
6	33	34 1/4	1 1/4	
4	22 5/8	30 1/4	7 5/8	
6	43 5/16	45 1/4	1 15/16	
5	37 5/8	37 1/4	....	3/8

It is seen that seven cases gained in weight during the fever, two showed a very slight loss and two an appreciable loss. The most remarkable cases occurred in a child of 3 years who showed a gain of 8 pounds after a fever lasting nineteen days, and another of 4 years gaining over 7 pounds after a fever of twenty days.

There were no very severe complications and no relapses. Under the former may be noted two cases of tonsillitis, two of otitis media, one hypostatic pneumonia, one pyelitis and one severe bronchitis.

Without a knowledge of the epidemic and the use of the Widal test, it is doubtful if most of these cases would have been properly diagnosed. In no case was the spleen palpable. They came in with the following diagnoses: Probable typhoid fever, 5 cases; Brill's disease, 1 case; meningitis, 1 case; miliary tuberculosis, 1 case; bronchitis, 1 case; no diagnosis, 2 cases.

I am indebted to Drs. Dennett and Steinmetz for aid in watching and collating the cases.

DIAGNOSIS OF WHOOPING-COUGH BY THE COMPLEMENT-DEVIATION TEST *

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CINCINNATI

The recognition of such a scourge as whooping-cough, particularly when occurring in institutions in which children are segregated in large numbers, is a matter of great importance.

In his address on this subject before this Society last year, Morse emphasized the difficulty—and the value—of early diagnosis.

By the time the paroxysmal stage is reached, the infection, where children are gathered together, is widespread, and the resulting mortality high, as shown by carefully collated statistics.

The desire to find a definite method of early diagnosis led us to a study of the complement-deviation test. With our modified technic we have been able to make the diagnosis of pertussis in all stages of the disease, catarrhal, paroxysmal and convalescent. Particular emphasis is laid on the point that the diagnosis can be made early in the catarrhal stage, long before any whoop appears, and at a time when prompt isolation of the infected child will prevent the spread of the disease. Thus several children admitted to the open children's ward of the Cincinnati Hospital with a diagnosis of bronchitis, gave a positive early reaction, though there was neither vomiting nor whoop. They were immediately removed to the pertussis ward, and our open ward remained free of pertussis all winter. A preliminary report of our work has already been published[1] from which the following excerpt is taken.

When Bordet and Gengou[2] described the bacillus of whooping-cough in 1906 they used the complement-deviation test to control their bacteriological findings. From this time on, it has been known that the blood of patients in the late stages of pertussis, during convalescence and for some time thereafter, would give a positive test showing definite deviation of complement. More recently it has been shown that in the later stages of atypical cases of pertussis, it is possible to deter-

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* Read at the meeting of the American Pediatric Society, Stockbridge, Mass., May, 1914.

1. Friedlander, A., and Wagner, E. A.: Jour. Am. Med. Assn., March 28, 1914, p. 1008.

2. Bordet and Gengou: Ann. de l'Inst. Pasteur, 1906, xx, 731.

mine the specific pertussis character of the infection by means of this test.[3]

Several observers have denied the possibility of making a diagnosis of pertussis even during the height of a typical attack or directly after convalescence by means of the complement-deviation test. Working in the Royal Serological Institute of Vienna, Bächer and Menschikoff[4] report that in twenty-seven cases of pertussis, moderate and severe, in the height of the attack, and in convalescence, attempts were made to obtain positive complement-deviation reactions, without success in a single case. Only after vaccines, prepared from pure cultures of the Bordet-Gengou bacillus were given was the test ever positive.

Commenting on these and other similar findings, Bordet[5] himself says: "I repeat that the power (of fixing the complement) is not seen early. In general it does not show itself markedly till toward the period of convalescence or cure." Netter and Weil[6] have reached practically the same conclusion. In fact it would appear to be the consensus of opinion that the test has no diagnostic value in the early stages.

Our own results lead us to the opinion that the complement-deviation test is of the greatest possible value in the diagnosis at all stages. Our results are herewith presented in tabular form.

TABLE 1.—RESULTS OF COMPLEMENT-DEVIATION TESTS FOR WHOOPING-COUGH

	No.	Positive	Negative	Per Cent.
Cases whooping	18	18	0	100
Normals	16	0	16	100
Early cases catarrhal stage	12	11	1	91.6
Not whooping; course otherwise typical	1	1	0	100

These cases were taken both from our hospital service and from private practice, and all cases were carefully followed.

It will be noted that the 18 cases tested during the paroxysmal stage gave positive reactions: 3 + in each case. Nine of the cases were in the first week of the whoop, and 3 early in the second week.

3. Bordet and Gengou: Centralbl. f. Bakteriol., Abt. I, Orig., 1911, lviii, 537; Bordet and Brunard: Bull. Acad. de méd. Belge, 1910, xxiv, 320; Delcourt: Presse méd. Belge, 1912, lxiv, 19.

4. Bächer and Menschikoff: Centralbl. f. Bakteriol., Abt. I, Orig., 1912, lxi, 218.

5. Bordet: Centralbl. f. Bakteriol., Abt. I, Orig., 1912, lxvi, 276.

6. Netter and Weil: Compt. rend. Soc. de biol., 1913, lxxiv, 236.

In no case did a normal give a positive reaction. This series included several cases of bronchitis. Of the 12 early cases, tested in the catarrhal stage, 11 gave positive reactions, and in each instance the definite whoop appeared later, thus confirming the serologic diagnosis clinically. The one case that gave a negative reaction was tested at the very beginning of the cough. The sister had given a positive test. Two weeks later the child with the negative test began to whoop.

TABLE 2.—TABLE OF ELEVEN POSITIVE REACTIONS TESTED IN THE CATARRHAL STAGE

No.	Duration of Cough Without Whoop	Time of Appearance of Whoop After Test
1	3 weeks	1 week
2	3 weeks	3 weeks
3	3 days	2 weeks
4	8 days	About 1 week
5	5 days	2 days
6	1 week	2 days
7	1 week	2 days
8	10 days	2 weeks
9	5 days	2 days
10	10 days	1 week
11	2 weeks	5 days

The one case giving a positive test that never whooped, occurred in a child with paroxysmal cough and vomiting lasting over a period of four weeks, evidently an atypical pertussis.

TECHNIC OF TEST

Our technic for the test has been as follows: A small amount of blood — about 15 to 20 drops — was taken from the patient's ear, finger or toe in small test-tubes or the Wright capillary tubes. For young children we have found the great toe very satisfactory.

The blood was kept at room temperature or placed in the incubator until coagulation had taken place. Serum was then separated more completely from the clot in the centrifuge. So far in our test we have used only fresh, active serum. Two drops of the serum were used in each test.

Hemolytic System: The Noguchi system was used because of its extreme delicacy and because of the small amounts of material — especially serum — required. In this system washed human corpuscles, 1 drop to 4 c.c. of salt water, are used.

Amboceptor: The amboceptor employed was prepared according to the Noguchi method, the serum being dried on filter paper. The complement was obtained in the usual way from guinea-pig — dilution 1 to 40. Aside from the delicacy of this hemolytic system, it is of great value in working with children because of the very small quantity of blood required. It is not necessary to take blood from the veins and the small quantity of blood required is easily obtained even from very young children.

Antigen: This is the most important factor in the test. The Bordet-Gengou bacillus was obtained in pure culture from the laboratories of Parke, Davis & Co. Most of our work was carried on with this culture. Cultures were

also obtained from the H. K. Mulford Co., and from Dr. F. B. Mallory of Boston. The latter culture came from a strain grown at Theobald Smith's laboratory.

Subcultures were made on Bordet's medium and an ascitic fluid agar. Latterly we have used the ascitic fluid agar exclusively and our antigens were made up as follows: Seventy-two-hour growths were taken. The colonies, which were very tenacious, were scraped off the agar with a glass hook into sterile salt water. An emulsion was made and the bacteria again washed in salt water. It is important to do the second washing so as to rid the emulsion of any particles of agar. From this washed emulsion a standard suspension was made, and 0.1 to 0.2 c.c. of this used in the tests. Throughout the test, live bacteria were used. It will be noted that we have always used fresh, active serum and live bacteria, and we consider this innovation of great importance.

Controls: In each test known normal and known positive controls were used. In each series of tests the hemolytic system was tried out in the usual manner, using a water bath at 37 C. for incubation. After primary incubation for half an hour, the amount of amboceptor indicated by the preliminary test was added to our final test-tubes and the tubes again incubated in the water-bath.

In every instance independent readings were taken by each of us, always without previous knowledge of the clinical history of the given case.

The test is not at all difficult to carry out for any one with experience in serologic work. Using the precautions above noted, the readings have always been sharp, clear and unmistakable. It is not difficult to keep the bacillus growing on the ascitic fluid agar, which for the purposes of the test is a much more satisfactory medium than the original Bordet.

The point of particular importance is that by this method and by this test the diagnosis of pertussis can be made with absolute certainty in the catarrhal stage. Finally it is a matter of record that the success of vaccine therapy depends in large measure on the time of its application. The earlier the vaccine is given the better the results. If it be possible to diagnosticate whooping-cough in the catarrhal stage surely and definitely, its rapid cure seems assured. We have just recently begun therapeutic vaccine work. In two cases—father and son—occurring in the private practice of one of us, the test was positive in the catarrhal stage in both instances. Both patients whooped subsequently, confirming the diagnosis clinically. Both of these cases were treated with a vaccine prepared from one of our own subcultures. The father received five injections at four-day intervals, 600,000,000 bacteria being given at a dose. In three weeks his cough had ceased completely. The boy also received five injections of same dosage, and in three and a half weeks his cough was over.

We believe, therefore, that the test as outlined is worthy of full confidence and that it offers definite early diagnosis and thus points the way to a rapid cure of pertussis.

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THE ARTIFICIAL COOLING OF SICK-ROOMS IN SUMMER

A PRELIMINARY REPORT *

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The mortality of gastro-enteritis in infants, despite all scientific efforts devoted to its amelioration, is so appalling, that each summer, in addition to employing what is best in the old methods of treatment, we attempt to devise newer and better ways of combating this formidable disease. It was with this in view that during my summer service of 1913, an endeavor was made to attack this malady in a way entirely different from any previously undertaken.

The etiology of gastro-enteritis has been and will be for years to come a much discussed subject. Without reiterating the well known conclusions of many pediatrists, based on theoretical considerations and practical observations, let us state that in our opinion also, gastro-enteritis is probably primarily a nutritional disorder, having as a secondary causal factor a bacteriologic basis. Both of these factors operate in winter as well as in summer. To account, then, for the marked prevalence of diarrheal diseases in summer we must consider a third potent cause, namely, the heat.

In order to determine the exact part heat really plays in the production of these summer diarrheas, and the effect of treatment in a routine manner with this important factor eliminated, a cooling plant was installed at the suggestion of Dr. Goldwater, superintendent of the hospital, in one small ward of the children's pavilion whereby the temperature of the room was kept constantly lower than that of the outside atmosphere. In brief, the method of cooling was as follows:

Outdoor air is forced by a motor-driven Sirocco fan through a water-chamber in which the water is cooled by brine coils. From this chamber the air is forced through an upper chamber filled with additional brine coils, thence through short ducts into the wards, where the cooled air enters near the floor level. Open transoms allow the warmer strata of vitiated air to

* Submitted for publication, May 27, 1914.

* From the Children's Service of Dr. Henry Koplik, Mt. Sinai Hospital, New York.

* Read at the meeting of the American Pediatric Society, Stockbridge, Mass., May 27, 1914.

escape from the top of the rooms. Some air also escapes through the entrance door when used by the nurse and the physician. The cooling plant proper is located in the basement under room No. 1, as shown on plan, Figure 1. It consists of a Greef air washer, a motor-driven blower and coils through which cold brine is forced, an eliminator for removing the free particles of moisture and heating coils for reheating the air, if so desired.[1]

The temperature was recorded by means of two recording thermometers, one registering the outdoor temperature, the other that of the room. The relative humidity was ascertained by a sling psychrometer. Observations of the temperature and relative humidity were also taken in the general ward where no cooling was done.

Typical charts of the recording thermometers are reproduced in Figure 2, showing the records for the hours from 9 a. m., July 31,

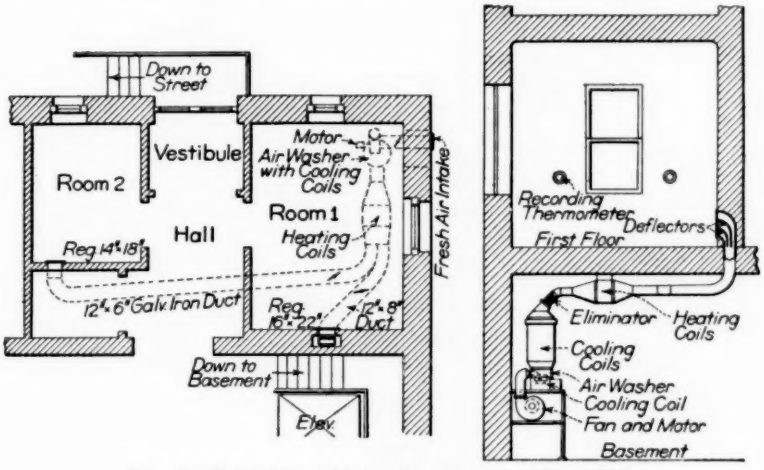

Fig. 1.—Location and details of air-cooling plant.

1913. The outdoor temperature, as will be noticed, was 86 F. at 9 a.m., rising gradually to an average of 92 between 1:30 and 4: p. m., then dropping gradually to 78 between 5 p. m. and 6:30 p. m., when it began to rise again. The indoor temperature of the room was maintained at an average of 72 between 9 a. m. and 3 p. m., rising gradually to 74 at 7 p. m., which latter temperature was maintained until midnight, then rising to 75 until 4 p. m., dropping again to 74 until 7 a. m. and 72 at 8:45 a. m. The temperature in the cooled room varied during the period of observation from 63 to 74. This latter temperature was reached only on one day when the temperature on the street rose to 93. The average temperature in the cold room for 252 readings was 69 during a period of forty-two days. The

1. From a description by A. M. Feldman, M. E., Consulting Engineer, in the Proceedings of the American Society of Heating and Ventilating Engineers.

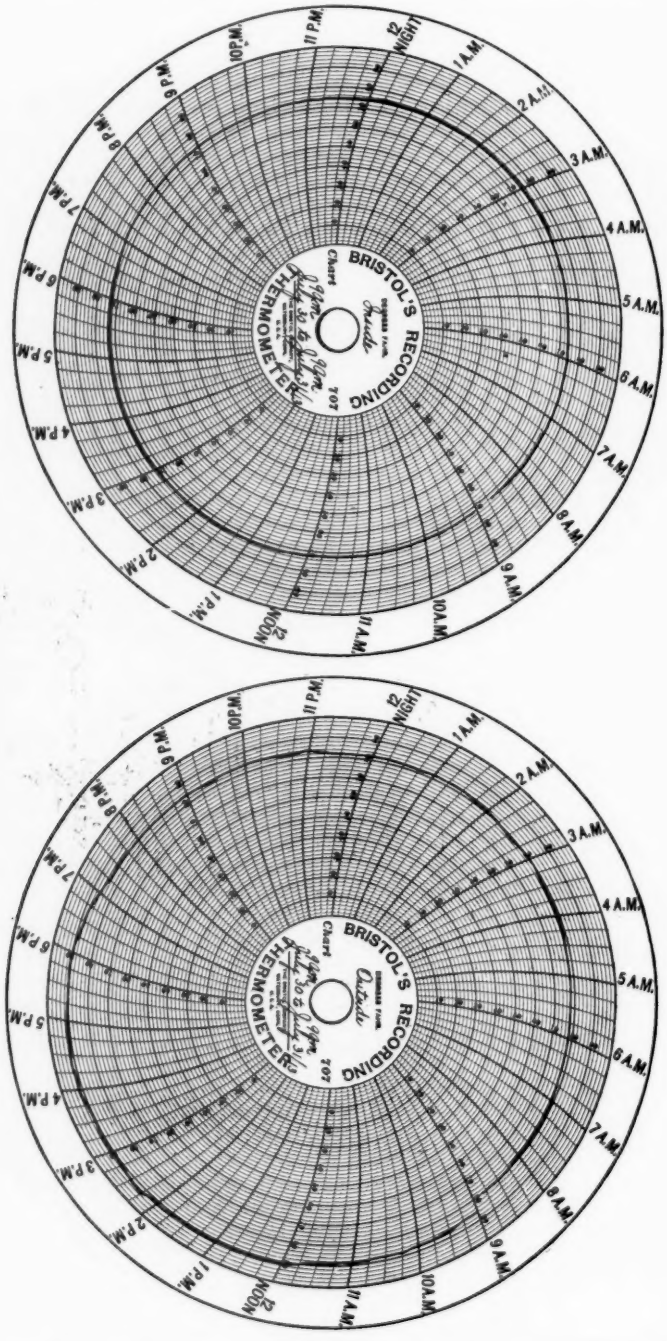

Fig. 2.—Charts showing record of inside and outside temperatures, July 30 and 31.

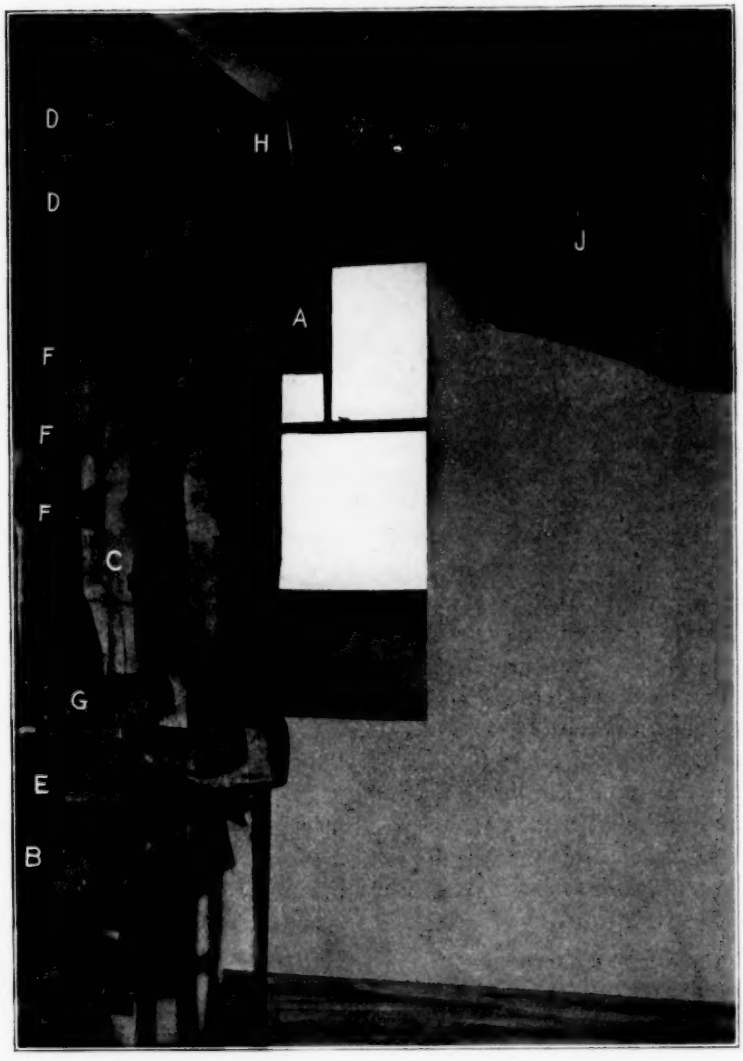

Fig. 3.—View of the air washing and cooling apparatus. The fresh air enters the duct at the point A in the upper sash of the window. At B may be seen the blower which delivers to the cooler-washer and cooling coils in chamber C. The pipes D D, are brine pipes tapping the return brine line of the general refrigerating system of the building. At E is the small pump for securing a positive circulation of the brine. The brine pump delivers to the washer through the pipe G and to the three coils above the washer F, F, F. At H is the delivery duct from the air washer and J is the reheating chamber, fitted, as indicated, with thermostatic control, the reheating coil also divided into three sections and the sections having hand valve control in the usual way. Incidentally the brine pipes are provided with thermometer wells and a thermometer is shown at the side of the duct to obtain readings of the air on delivery from the air-washer cooling system. A thermometer was also arranged for on the delivery side of the reheating chamber, so that arrangements are available for making a comprehensive test.

records of the outdoor temperature during the same forty-two days, taken at 8 a. m., 12 m., 4 p. m., 8 p. m., 12 p. m. and 4 a. m., were 77, 79, 79, 80, 74 and 70 F., respectively. The temperature of the cold room was at all times from 5 to 11 degrees below that of the outside air.

The relative humidity was observed at different intervals during the day. In the artificially cooled room, the relative humidity varied between 41 and 76, making an average of 60 for readings taken on twenty-five different days. In the main ward, the relative humidity varied during the same period from 35 to 80, with an average of 59.

It is interesting to note that while low temperatures were obtained in the cold room, the relative humidity was approximately the same percentage as that in the main ward with higher temperature, thus indicating that the absolute humidity was reduced by the process of cooling.

Thirteen selected cases were treated, the average period of treatment being about two weeks. By selected cases is meant patients suffering from gastro-enteritis, ruling out premature and moribund infants. Six of the thirteen were in poor condition at the time of admission. One of these died shortly after entering the hospital. All the others were discharged from the hospital well. The treatment in all these cases was not different from that ordinarily carried out; this included *Eiweissmilch,* skimmed milk or barley water, plus the usual symptomatic medication.

Inasmuch as only thirteen cases of gastro-intestinal diseases were treated in the artificially cooled ward, it is hardly justifiable to present definite conclusions as to the therapeutic value of this measure. Gastro-enteritis was not particularly prevalent during the summer of 1913, and the number of cases treated in the hospital was very much below the number usually treated; in fact, the thirteen patients who were treated in the cooled ward were practically all the gastro-enteritis cases available for our first tentative studies. Thus there was no opportunity for comparison of the behavior of these cases with that of patients treated for the same disease in the open wards, under ordinary conditions. While unable at the present time to draw far-reaching conclusions, we have nevertheless gained the impression that the babies treated in this ward were, on the whole, more comfortable, cried less often, looked less sick and less parched, rested and slept better than the patients kept in the other wards of the children's service during the same time.

It is not our intention to belittle the present methods of treatment of this disease, as well as the efforts of the Board of Health, the Social Welfare Department, milk depots and other associations for

the prevention of infant mortality, but simply to introduce this additional therapeutic measure as a valuable adjuvant in the eradication of this malady. The only cases in which this method may be harmful are in weaklings, premature and moribund infants, in whose cases extreme heat is a powerful factor in sustaining life. In all other cases of gastro-enteritis, no matter how severe, the artificial cooling of the sick-room will in our opinion prove to be an extremely helpful measure.

I hope to have the opportunity of studying this important question further this summer. I wish to thank Dr. Louis H. Levy and Dr. A. Brody for their kind assistance in carrying out these observations.

30 West Eighty-Eighth Street.

CIRCUMCISION IN THE MASTURBATION OF FEMALE INFANTS*

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NEW YORK

The masturbation of female infants while really not common occurs with moderate frequency. At the meeting of this society in 1907, Dr. B. K. Ratchford in his presidential address reported fifty-two cases of masturbation in infancy observed by members of this society, only four of which occurred in male infants.

This habit, if neglected, leads to a considerable amount of moral depravity, and on the other hand, it may be fairly controlled by proper treatment. It is, therefore, a serious condition which is fairly controllable and one to which too little attention has been devoted. I say nothing of the masturbation of male infants, for sufficient attention is paid to the hygiene of the male sexual organs so that the sort of irritation which leads to masturbation in infants rarely e boy babies. On the other hand, in the female infant there exist fairly regularly marked adhesions between the sensitive clitoris and the surrounding tissues so that on examination the clitoris is frequently found to be buried in these adhesions.

In a normal child these adhesions seem to cause little irritation; but in the nervous, sensitive child they may cause intense irritation and lead to the formation of a habit which, if untreated, may become permanent and exert a most injurious influence over the future development of the child.

The treatment of this condition is both general and local. The general health should be improved by such hygienic measures as are available, and all sources of nervous irritation and excitement should be, as far as possible, removed; but no cure can ordinarily be accomplished by such measures alone. The common use of an apparatus for separating the thighs is of little avail, and the only curative treatment is that applied to the removal of the active source of irritation, the adhesions of the clitoris. These may be separated without the use of an anesthetic. The operation is very painful, and such separation under these conditions is difficult and apt to be followed by the formation of other adhesions. The only satisfactory method of treat-

* Submitted for publication, June 13, 1914.
* Read at the twenty-sixth annual meeting of the American Pediatric Society, at Stockbridge, Mass., May 26, 1914.

ing this condition is by circumcision, an operation which should be performed by one who is accustomed to do it, the foreskin being removed as completely as possible.

The result of this operation is most satisfactory. In many cases no recurrence of the habit is noticed after the operation, while in others recurrences will occur; but they are found to be due to adhesions which have formed notwithstanding the circumcision.

The existence of so serious a complication in a young infant long before the development of the sexual activity leads one to wonder whether more attention should not be paid to the condition about the clitoris in all young girls with symptoms of nervous irritability. It has been said that the clitoris is an electric button which rings up the whole nervous system. It is my belief that more attention should be paid to the hygiene of this region.

The histories which one obtains of these cases are not always clear. The common history of a child putting its fingers on the genitals at every opportunity, while showing evidence of some irritation, is naturally not sufficient for a diagnosis of masturbation. The real condition is often misunderstood by the parents, but when masturbation exists, a clear history of an orgasm can be obtained after close observation on the part of the attendants.

A few illustrative cases of this sort I will enumerate.

CASE 1.—A female infant aged 10 months was noticed frequently to rub her thighs actively for a certain period and then to become quiet, breaking into a perspiration. This habit had been noticed for several weeks and was becoming more frequent. Examination showed the clitoris to be entirely buried in adhesions. These were somewhat broken down and the clitoris moderately cleared without, however, any effect on the habit, so that an operation was advised. A complete circumcision was done two months ago and at the same time a large adenoid was removed. Since that time no masturbation has been noticed although carefully watched for.

CASE 2.—A girl aged 3 years had probably from the history been masturbating for more than a year, this habit having become worse, especially at bedtime and in the morning. The real character of this habit had only just become evident. The child was circumcised under chloroform anesthesia and at the same time a large adenoid was removed. More than two years have elapsed since this operation without any recurrence of the habit, although a year after the operation the child had an attack of chorea which lasted for about a month.

CASE 3.—A child aged 5 years had weighed only 5 pounds at birth, but had done well until she was 8 months old, when she contracted a colitis followed by the condition known as the infantilism of Herter, from which, however, she recovered again in the course of a year. She is a frail, nervous child, and it has only recently been noticed that she masturbated. A partial separation of the adhesions without an anesthetic did not remove the habit, so a circumcision was performed under anesthesia, and she has never been known to masturbate since, although five months have elapsed since the operation.

Other cases have shown less favorable results from the operation.

CASE 4.—A child aged 5 years, frail and nervous, with red hair but usually well, was found to masturbate. A circumcision was done under anesthesia fol-

lowed for a time by a cessation of habit; but three months later the habit was again noticed and on examination new adhesions were found to have formed, and the breaking down of these was followed again by a cessation of the habit for six months. Since that time there have been several periods when the habit was resumed, always associated with adhesions about the clitoris. These, however, have become less frequent and now there is no history of a recurrence during the past year, five years after the operation.

CASE 5.—A child aged 18 months with a cerebral palsy and poor intellectual development gave a history of straining which was supposed to be associated with constipation. She was a premature child weighing 3 pounds at birth who had convulsions on the fourth day. She did not talk or stand, and both the right leg and right arm were weak and of little use. She was said to strain when lying down, would get red in the face, perspire, burrow her head in a pillow and then go to sleep. The clitoris was found to be buried in adhesions and a circumcision was performed. Two months after the operation there were still some slight attempts at masturbation and three months later there were almost none. Five months later it was reported that she had been free from attacks, but for a week past these had recurred, and on examination it was found that new adhesions had formed. She was much better after these were broken down. Six months after the operation she was brought to New York on account of her renewal of the habit, and the clitoris was found fairly buried in adhesions. These were broken down without an anesthetic. This child left four years ago for Australia and it has been impossible since then to keep in close touch with her condition, although the father writes me that she has entirely ceased straining. Here was a difficult case on account not only of the recurrence of the adhesions but also of her mental deficiency.

CASE 6.—Another case very resistant to treatment but always benefited by the breaking down of adhesions was a child I first saw ten years ago who was then 10 months old. She was frail, nervous and had red hair. When only 10 days old what was apparently a typical masturbation was noticed. She was treated with braces, which seemed to control the habit temporarily. She had a suspicious eruption during the first year but never any clear evidence of syphilis. She had a typical orgasm in my office, covering her face with her skirts and rubbing her thighs together, taking a number of deep inspirations and then breaking into a sweat. The clitoris was found bound down by adhesions and a circumcision was performed, after which she was better. Seven months after the operation the mother reported that there had been an entire cessation of the habit until two weeks before. The clitoris was found again buried in adhesions, and a second circumcision was performed under an anesthetic. Since that time there has been a steady gradual improvement in her condition. Occasionally new adhesions have been broken down, but without an anesthetic, and periods of months have elapsed with no attempt at masturbation and there has been no history of the habit now for three years.

CONCLUSION

The masturbation of female infants occurs in nervous children, the stimulating cause being adhesions about the clitoris such as exist in practically all female infants.

The treatment of this condition by braces alone is palliative and not curative. The only curative treatment is by separation of the adhesions and circumcision, and the result of such operative interference is often satisfactory.

211 West Fifty-Seventh Street.

MIDDLE EAR COMPLICATIONS OF MEASLES IN IMMIGRANT CHILDREN *

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The occurrence of middle ear suppuration as a complication of measles has been recognized, but the extreme prevalence of this complication has not been realized. In fact, so far as I know, there never have been any real data produced in any of the works by authorities on the contagious disease, as to the incidence of middle ear disease in measles.

The following, taken from the records and statistics of the Contagious Disease Hospital at Ellis Island, will give some idea of the extent and nature of the disease as met with in immigrant children, and may indicate to some extent its occurrence in all cases. Of course the conditions met with in Ellis Island differ greatly from ordinary cases in general practice, not only in the class of patients, but in the stage of disease and in the care the patients have had previously on board ship; and undoubtedly the percentage of all complications is higher than ordinary.

First of all, I would like to do away with the idea as held by some people, that the form of measles found in foreign children differs from that found in the native born. True measles is the same irrespective of country or nationality.

Middle ear suppuration is by all means the most common complication met with in all cases. In infants less than 18 months old it ranks second only to bronchopneumonia, and the reason of this may lie in the conditions of life on board ship.

From the time the hospital was opened in June, 1911, until Dec. 1, 1913, there were admitted in all 1,769 cases of measles. The average age of all patients was 4.81 years; and of these 321 cases, or 18.2 per cent., developed middle ear disease.

In addition to these, several cases proved to be diphtheria carriers, and these developed middle ear disease, which showed Klebs-Loeffler bacilli in the discharge and proved extremely obstinate.

The length of time an uncomplicated case of measles remained in the hospital varied from seven to thirty days, accounted for by the

* Submitted for publication, April 3, 1914.

degree of disease on admission. The average time of all cases was approximately two weeks. Those cases developing ear trouble (exclusive of mastoid involvement) remained on an average for 45.12 days.

Bacteriologic examination of the discharge from the ears gave a preponderance of *Staphylococcus aureus*. In some a bacillus (probably Hoffman's) was found. In those cases in which a streptococcus appeared, the child was much sicker and more resistant to treatment. This last holds also of the diphtheria infections, as shown by the records of two children who developed mastoids with a stay in the hospital of 213 and 241 days, respectively. Even then they were not entirely well.

It is interesting to note the comparatively low incidence of mastoid involvement. Out of the whole 321 cases, only twenty-nine, or 1.6 per cent., developed this troublesome feature, with an average stay in the hospital of 75.64 days.

The onset of middle ear suppuration occurred most frequently after the measles rash had disappeared, and usually several days after admission. In a few cases it was present on admission. In nearly all cases it appeared suddenly and with little or no pain. In very few cases did we see acute otitis and paracentesis of the drum was a rare event. Moreover, little pain was experienced during the progress of the disease. Usually, but by no means always, was there an elevation of temperature for twenty-four to thirty-six hours before the suppuration occurred. In general the process began and persisted as subacute.

The treatment found most effective consisted in keeping the middle ear clean. This was accomplished by frequent and copious irrigations at low pressure. The solution found to be most beneficial was 1 to 10,000 mercuric chlorid made up in normal salt solution. About a pint was used of this for each ear three or four times daily with the fountain syringe at a height of about two feet above the ear. After each irrigation the canal was wiped dry with sterile cotton.

The most obstinate period of the treatment was at the last when there was very little discharge. This would often resist treatment for days, and as we made it a rule not to discharge a patient until the ear had been dry for two days, the length of stay in the hospital was often materially increased. Very often the convalescence was hastened by instilling drops of alcohol and mercuric chlorid after each irrigation.

The ultimate results of course cannot be determined, as it is impossible to follow up these children; but in the majority of cases the hearing of the children on leaving the hospital was good; the drum membrane more or less completely restored.

To sum up then, suppurative otitis media occurs as a complication of measles in immigrant children; that is to say, in children subject to

unfavorable hygienic surroundings both at home and on board ship, in nearly 20 per cent. of cases. Mastoid involvement is not common, and the disease yields to treatment comparatively readily. The disease is essentially subacute (rather than chronic) and the apparent results as affects hearing are good.

A CASE OF TUBERCULOUS MENINGITIS COMPLICATED BY INFLUENZAL MENINGITIS *

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NEW YORK

Tuberculous meningitis complicated by purulent meningitis is a rare event. In a search through the literature I have been able to find records of twenty-one cases only. Paisseau and Tixier[1] reviewed the subject in 1909 and collected nine cases, adding one of their own. They, however, overlooked six cases reported, respectively, by Heiman,[2] Lenhartz,[3] Heubner[4] (three cases) and Kneass and Sailer.[5] Since 1909 two more cases have been reported by Guinon and Grenet,[6] and Dupérié.[7] To these should be added three cases from the records of the Babies' Hospital and the one here presented in detail.

The organism found in these instances of mixed infection were the meningococcus, the pneumococcus, the streptococcus, *M. tetragenus,* the colon bacillus and an unclassified Gram-negative diplococcus growing freely on all media. In no case was *B. influenzae* found. Nine of the twenty-one cases were due to the meningococcus.

The fact that influenzal meningitis associated with tuberculous meningitis has not hitherto been described is a somewhat surprising fact, for influenza has long been known as a particularly frequent and severe complication of tuberculosis. Pfeiffer in his report[8] of the successful cultivation of the influenza bacillus called attention to the fact that the mortality of patients with pulmonary tuberculosis was very high during the influenza pandemics of 1891 and 1892. From the sputum of such patients he was able in a number of instances to

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* Submitted for publication, May 13, 1914.

1. Paisseau, G., and Tixier, L.: Méningite et surinfection, Gaz. d. hôp., 1909, lxxxii, 979.

2. Heiman, Henry: Tubercular Meningitis, Arch. Pediat., 1897, xiv, No. 2.

3. Lenhartz: Weitere Erfahrungen über die Lombalpunction, Deutsch. med. Wchnschr., Vereins Beilage, 1897, p. 130.

4. Heubner: Ueber den Meningococcus, Deutsch. med. Wchnschr, Vereins Beilage, 1897, p. 109.

5. Kneass and Sailer: A Case of Tuberculous Meningitis with Secondary Infection, Jour. Nerv. and Ment. Dis., 1904, xxxi, 660.

6. Guinon, L., and Grenet, H.: Méningite à méningocoques et à bacilles de Koch associés, Rev. internat. de tuberc., 1912, xxi, 33.

7. Dupérié, R.: Gommes tuberculeuses hypodermiques multiples chez un nourrisson. Méningite terminale à méningocoques et à bacilles de Koch, Arch. de méd. des Enf., 1912, xv, 599.

8. Pfeiffer, R.: Die Aetiologie der Influenza, Ztschr. f. Hyg., 1893, xiii.

isolate the influenza bacillus. Petruschky,[9] in a study of the secondary infections of pulmonary tuberculosis, found that next to streptococcus infection influenza was the most common complication. Similar conclusions, based on cultures from the sputum and from the lungs at autopsy, were reached by Kirchensteiner,[10] Schabad,[11] Klieneberger,[12] Kruse[13] and Wohlwill.[14] Wohlwill, in a very careful study of tuberculous lungs at autopsy, found that by making cultures from the finer bronchi he could isolate *B. influenzae* from sixteen of the seventy-three cases he examined, or from 22 per cent. Even more striking are the results obtained at the Babies' Hospital during the last three years. Cultures from the lungs have been made in fifty-four cases of pulmonary and general miliary tuberculosis and in these the influenza bacillus was found twenty-three times, or in 42.5 per cent.

In view of these facts it is perhaps surprising that no case like the present one should have been reported before.

AUTHOR'S CASE

The patient whose history is the subject of this report was admitted to the Babies' Hospital, Feb. 3, 1914, for convulsions. He was an Italian child, 5 months old. For the previous two months the mother had had a cough and this was the only suggestion of exposure to tuberculosis that could be obtained. Two weeks before admission, for a reason not given, the baby was weaned and since then had not thrived, though no other untoward symptoms had been noticed. Three days before admission the patient had a general convulsion lasting several minutes, after which the head became retracted and remained so ever since. Between the onset and the time of admission to the hospital he had about twenty more convulsions, and in the intervals was restless and irritable and slept little. He did not vomit.

On admission there was seen an irritable, poorly nourished child who looked acutely ill. The head was retracted and the neck was very stiff. The fontanelle, though slightly bulging, was not tense. The only abnormal signs in the chest were a few coarse râles at the angle of the right scapula. The liver and spleen were felt just below the costal margin. The legs were semi-flexed and slightly spastic and the knee-jerks exaggerated, but Kernig's sign was not present.

Examination of the blood showed 19,000 leukocytes, of which 35 per cent. were polymorphonuclear and 65 per cent. were lymphocytes. Lumbar puncture (February 4) showed 15 c.c. of clear fluid under normal pressure. In this there were 145 cells per c.mm., 92 per cent. of which were lymphocytes. No microorganisms were found in the smear.

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9. Petruschky: Zur Behandlung fiebernder Phthisikern, Charité Ann., 1892-3, xvii and xviii.

10. Kirschensteiner: Studien zur Bacteriologie der Lungen und Bronchialeiterungen, Deutsch. Arch. f. klin. Med., lxxv.

11. Schabad: Die Mischinfection bei Lungentuberculose, Ztschr. f. klin. Med., 1897, xxxiii.

12. Klieneberger: Ueber hämoglobinophile Bazillen bei Lungenkrankheiten, Deutsch. Arch. f. klin. Med., 1906, lxxxvii.

13. Kruse: Zur Aetiologie und Diagnose der Influenza, Deutsch. med. Wchnschr., 1894.

14. Wohlwill: Ueber Influenzabazillen im Bronchialbaum, Münch. med. Wchnschr., 1908, lv, No. 7.

The Von Pirquet skin test was strongly positive.

A second lumbar puncture was done the next day and showed a fluid having the same general characteristics as the first. A smear from it showed, however, a number of short, slender, Gram-negative bacilli which were morphologically identical with *B. influenzae*. An attempt to grow these on blood-agar failed.

A blood-culture taken at this time showed a profuse growth of two organisms, *Staphylococcus albus* and *B. influenzae*. The former was regarded as a contamination from the skin; the significance of the latter was not apparent.

February 6 a choroidal tubercle was discovered in the right fundus and on February 14 tubercle bacilli were found in the spinal fluid. The fluid though still perfectly clear was now under increased pressure, contained 375 cells, practically all lymphocytes, to the c.mm. and formed a film after standing twelve hours.

These findings established the diagnosis of tuberculous meningitis and inclined us to regard the presence of other organisms in the blood and spinal fluid as accidental. The only evidence from the spinal fluid of a cerebrospinal infection other than tuberculosis was the presence of influenza-like organisms in a single smear and this was not corroborated by culture.

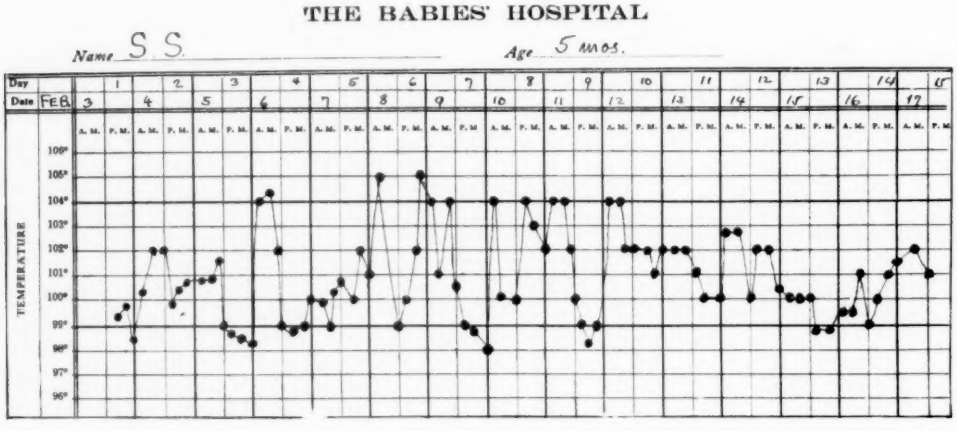

Chart showing long-continued, intermittent type of temperature in author's case.

Nevertheless, the clinical symptoms were very different from those of uncomplicated tuberculous meningitis. From the time of admission to that of death the patient had almost continual general clonic convulsions. The attitude was most striking. The head was retracted, the back was curved in extreme extension, the legs were flexed on the thighs and the thighs were flexed on the trunk. The muscles were all spastic. Frequent facial twitchings were observed and there was nystagmus. In view of the autopsy findings it is worth noting that no unilateral motor symptoms were ever noted.

The temperature, in marked contrast to that usually seen in tuberculous meningitis, ran a high and irregular course. So many similar temperatures at this season had been found in this hospital to be due to influenza that a culture was taken from the bronchial secretion and in this *B. influenzae* was demonstrated.

About ten days after admission signs of partial consolidation were found in the left upper lobe. Whether this was due to tuberculosis or to an intercurrent broncho-pneumonia was not clear.

The patient died from exhaustion sixteen days after admission.

The autopsy showed glandular, pulmonary and general miliary tuberculosis. The oldest lesions were in the left bronchial lymph-nodes, which were matted together round the main bronchus and in the hilum of the lung. In the left upper lobe were a number of caseous areas and a walnut-sized cavity just under the pleura. Surrounding this cavity was a zone of broncho-pneumonia.

The brain showed two distinct processes. The pia everywhere was studded with small miliary tubercles and there was at the base a fibrino-purulent exudate such as is usually seen in tuberculous meningitis. There was also present over the convexity of the left hemisphere an abundant, thick grayish-yellow, purulent exudate. This was not present on the right side.

The left middle ear was normal but the right contained a small quantity of thick pus.

Smears and cultures from the pus on the left convexity showed *B. influenzae* only, while a smear from the right ear showed both influenza bacilli and pneumococci. A culture from the bronchopneumonic area in the left upper lobe also showed a pure growth of *B. influenzae*. The strain recovered from the brain was injected into a rabbit and a guinea-pig and proved fatal for both within twenty-four hours.

Analysis of the clinical symptoms and the laboratory findings in this case reveals two striking points of difference from the picture usually seen in tuberculous meningitis; namely, the evidence of extreme cortical irritation and the long-continued, intermittent type of temperature (see chart). It was thought that the temperature might be explained by the respiratory influenzal infection, but the signs of cortical irritation, though frequently commented on during life, were adequately explained only by the autopsy. Considering the comparatively low leukocyte count and the lymphocytosis and the character of the cerebrospinal fluid, it was impossible to make a diagnosis of purulent meningitis during life.

The portal of entry for the influenzal infection in this case was evidently the respiratory tract, whence we may assume that it entered the blood-stream and then lodged in the meninges.

Why the cerebrospinal fluid failed to give any evidence of suppuration is difficult to explain. Several hypotheses occur to one, but perhaps none is necessary. In a case of meningitis reported by Dr. Josephine Hemenway[15] nothing abnormal was found in the spinal fluid before death, yet at autopsy there was discovered over both frontal lobes a purulent exudate from which the pneumococcus was grown in pure culture. This case demonstrated beyond a doubt that the products of meningeal inflammation do not of necessity migrate into the cerebrospinal fluid.

In conclusion, I wish to thank Dr. L. Emmett Holt for permission to publish this case, and Dr. Martha Wollstein for assistance in the pathological work.

Babies' Hospital.

15. Hemenway: Two Cases of Acute Meningitis of the Convexity, Arch. Pediat., March, 1908.

PROGRESS IN PEDIATRICS

ACTIVE IMMUNIZATION AGAINST DIPHTHERIA BY MEANS OF VON BEHRING'S VACCINE, AND THE DIPHTHERIA TOXIN SKIN REACTION *

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I. ACTIVE IMMUNIZATION

The weak point in the immunizing use of diphtheria antitoxin lies in the short duration of the passive immunity thus conferred. The injected antitoxin is rapidly broken down and eliminated until in the average case there is not sufficient antitoxin left at the end of ten days to afford further protection. (Von Behring, Park.) While this ten day period may be enough to protect the average individual who has been in contact with a case of diphtheria, it is not adapted to protect those who are constantly or repeatedly exposed to infection, as for example nurses or physicians on service in a diphtheria ward, or patients in the wards of a children's hospital. The rapid destruction and elimination of antitoxin is due in large part to the use of a serum obtained from an animal of a foreign species. Park found that if a guinea-pig received 10 units of diphtheria antitoxin obtained from a horse (heterologous serum) there was only ½ unit left at the end of seven days and 1/20 of a unit at the end of fourteen. When, on the other hand, a guinea-pig was given 10 units of guinea-pig antitoxin (homologous serum) there was 1 unit left at the end of fourteen days. Matthes gave a 4 kg. child 350 units of an homologous serum. Following the injection the content of the blood serum in antitoxin reached a height of 1/5 units per c.c., and one month later 1/20 of a unit per c.c. could be demonstrated.

In practically every non-fatal case of diphtheria there is an active production of antitoxin as a result of the reaction of the body to the absorbed diphtheria toxin, which persists for a varying length of time. Park mentions a case of diphtheria in which the blood showed 60 units of antitoxin per c.c. at the end of the fifth day, 90 per cent. of which was produced by the patient. The average case forms very much less than this. In this connection the findings of Kissling are most interesting. Kissling studied the production of antitoxin in physicians and nurses working in diphtheria wards. Those who had never had clinical diphtheria but who had been working about the wards for a long time

* Submitted for publication, May 1, 1914.

showed high amounts of antitoxin in their blood serum, while those recently assigned to duty showed low amounts and frequently contracted the disease. In other words an insensible active immunization had gradually taken place in those who had been on duty in the diphtheria wards for some length of time. The recent work of Schick and his coworkers would seem to show that active immunization is an exceedingly common occurrence, and that a large percentage of individuals—although they may never have had clinical diphtheria—possess sufficient antitoxin to protect themselves against infection except probably during severe epidemics.

The idea of the production of active immunization for purposes of prophylaxis is by no means new, and has been suggested by a number of workers in this field in past years. In 1909 Theobald Smith reported that he had obtained an active immunization in guinea-pigs, which lasted over several years, by the injection of toxin-antitoxin mixtures. Mixtures containing a slight excess of toxin produced the best immunity. Smith at this time suggested the use of toxin-antitoxin mixtures in man, and recognized the limitations of such a method. He found that diphtheria toxin alone produced a very slight degree of immunity with severe local lesions. Later, together with Brown, he reported the production of immunity in guinea-pigs by the injection of toxin in amounts so small that local necrosis did not occur. The immunity obtained in this way was sufficient to enable the guinea-pig to withstand one-half the lethal dose without a local reaction.

The first attempt to produce active immunization in man by means of toxin-antitoxin mixtures was reported at the 1913 meeting of the Kongress für Innere Medizin at Wiesbaden by Hahn, who presented the results of the use of such a vaccine prepared by von Behring at Marburg, and during the past year a number of papers on this subject have been published by von Behring and his associates. While the idea of active immunization by toxin-antitoxin mixtures is not new, as we have shown, von Behring claims originality through the results of his studies with such mixtures. It is not within the scope of this review to enter into a discussion of the relationship and combination of toxin and antitoxin, and we simply state von Behring's claim of the reversibility of such combinations. He found that a mixture in which 1 unit of toxin was neutralized by 1 unit of antitoxin for the guinea-pig, was not necessarily atoxic for other species of animals. Thus if 1 unit of toxin is combined with 20 to 40 units of antitoxin and the mixture injected into a monkey, the toxin causes death, and it is necessary to use 80 to 100 guinea-pig antitoxin units to neutralize 1 unit of toxin for the monkey.

The so-called "von Behring's vaccine" consists of a mixture of strong diphtheria toxin and antitoxin in such proportions that the toxin

is just neutralized, or is in very slight excess, when tested on a guinea-pig. A number of different mixtures have been used which are designated as M 1/M 11, MM 1, etc., and which vary somewhat in their proportions. The mixtures remain active for ten weeks after preparation. At first the vaccine was injected subcutaneously and the formation of antitoxin in the blood serum studied by Römer's method. It was found that from twenty to twenty-five days elapsed before the development of immunity.[1] It was later found that if the injections were given intracutaneously a stronger immunity resulted and the interval between the injection and the development of immunity was reduced to ten days. This largely overcomes the most serious practical objections to the use of the vaccine.

The vaccine produces a reaction at the site of injection which is similar to the local tuberculin reaction. By testing the blood of vaccinated individuals it has been found that the degree of the local reaction corresponds to the amount of antitoxin formed, and hence furnishes a simple and reliable index of the degree of immunization produced. It is impossible to test the blood for its antitoxin content where a number of people are vaccinated, and Hahn and Summer have put this phenomenon to practical use by recording the degree of immunization according to the degree of the local reaction, and have elaborated a scheme for recording the local reaction according to the area and degree of reddening, infiltration, and scaling that occurs. According to Kleinschmidt and Viereck the local action of the toxin of the vaccine does not depend on the antitoxin in the blood, as is the case when diphtheria toxin alone is injected intracutaneously. A negative reaction with the latter is evidence of antitoxin in the blood, and it has frequently been observed that individuals with antitoxin in the blood are more susceptible to the vaccine than those without. Antitoxin in the blood—which depends on an active immunization from previous infection and which is found in some 50 per cent. of people—apparently leads to a hypersensitiveness toward the toxic agent. Kratz has shown that a toxin-antitoxin mixture which is toxic for an actively immunized animal is neutral for a control animal of the same species. It is suggested that a previous active immunization leads to a more rapid cleavage of the toxin-antitoxin mixture.

Hahn in his original report gave the results of the use of the vaccine in forty cases. No bad results were noted. The formation of antitoxin was studied in all of these cases which are largely

1. The antitoxin content of the blood serum which affords immunity apparently lies between 1/10 and 1/30 units per c.c., except perhaps in case of infection with organisms of high virulence.

of theoretical interest. Matthes likewise studied the formation of antitoxin. The results in one case (K) were as follows:

December 23, K. was given 0.25 c.c. of MM 1/4 subcutaneously. Previous to vaccine injection the blood showed an antitoxin content of ⅛ units per c.c. Later tests were as follows:

10 days after injection — 160 units of antitoxin per c.c.
16 days after injection —175 units of antitoxin per c.c.
21 days after injection — 20 units of antitoxin per c.c.
12 weeks after injection — 8 units of antitoxin per c.c.

(v. Behring reckons that it will be two years before antitoxin falls to 1/100 units per c.c., in this case.)

Zangmeister reported that new-born children do not react to large doses of the vaccine. Kleinschmidt and Viereck studied the antitoxin formation in twenty-seven children and came to the conclusion noted above, that intracutaneous injections are more active and that the degree and rapidity of immunization corresponds to the strength of the local reaction.

Following these rather theoretical studies, in which no bad results were observed, Kissling, and Hahn and Summer, used the vaccine in a large number of cases in order to test its practical value. Kissling injected 310 patients in the wards of his hospital at Hamburg who had been exposed to diphtheria. Of these 111 received two injections and no cases of diphtheria developed after vaccination. One hundred and ninety-nine were injected once and eight of these subsequently developed clinical diphtheria. The cases which developed were in the scarlet fever and pertussis wards.

Scarlet fever: 197 patients were immunized; 108 injected twice; 89 injected once; 5 cases developed.

Of these 1 developed on the fourth day after injection; 1 on fifth day after injection; 1 on nineteenth day after injection; 1 on thirty-fifth day after injection; 1 on thirty-seventh day after injection. The last three were very mild.

Pertussis: Thirty cases immunized; three cases of diphtheria developed.

Kissling observed four severe reactions when the vaccine was injected into the arm and prefers to inject into the skin of the back along the line of the scapula.

Hahn and Summer tried out the vaccine in certain villages and small cities where diphtheria was epidemic. They found a tremendous difference in the susceptibility of different individuals to the vaccine, some reacting less than others to doses fifty times greater. For this reason they first inject a small dose (0.01 c.c. intracutaneously) and if a severe reaction is obtained a second injection is not given, as the first was found to give satisfactory immunity. If a mild reaction is obtained, a second injection with a doubled dose is given on the third day, and if no reaction followed the first injection a 10-times dose is given at the second injection. They advise two or even three

injections a day apart unless the first or second reactions are very severe. Children were vaccinated in five villages and cities and as many did not return for examination after the first or second injections, the cases are divided as follows:

1. Complete immunization: good repeated reaction.

2. Doubtful immunization: slight reaction after first and did not return after second injection.

3. Unsatisfactory immunization: did not return for examination after first injection.

An example of their results is as follows:

CITY OF EGELN (5,300 INHABITANTS)

Cases from January 12 to November 11..... =72
First immunization, November 11..... } 7.5 per cent. mortality.
Cases November 11 to December 18..... =27

	Total No. Children	Cases of Diphtheria which Developed after November 11
Non-immunized	1,089	21
Inside first ten days.....	....	4
Unsatisfactory immunization	89	0
Doubtful	130	1
Complete	304	1

Thus 20 per cent. of the non-immunized developed diphtheria and 10 per cent. of the immunized. If the four cases developing in the first ten days are omitted, the percentage of immunized cases is only 3.3. Of these four cases, one was so mild that antitoxin was not used. The totals were as follows:

	Total No. Children	Cases of Diphtheria
Complete immunization	633	2
Doubtful immunization	255	2
Unsatisfactory immunization	209	1
Cases developing in first ten days after injection.....		10
Cases developing among non-immunized.....		51

Of the two cases developing among the "completely immunized," one was abortive and the other was a clinical case without bacteriological control.

A "negative phase" between the time of the injection and the development of immunity has not been noticed by any who have used the vaccine so far. On the contrary Hahn and Summer note that the ten cases of their series which developed within ten days after injection were all characterized by a mild abortive course. The antitoxin portion of the vaccine has not produced an hypersensitiveness to further injections of antitoxin used for curative purposes in those patients who developed diphtheria subsequent to the injection of the toxin-antitoxin mixture.

The vaccine is still in an experimental stage and its final value and usefulness remain to be determined. So far no bad or unpleasant results have been reported. If further use supports these early impressions the vaccine will probably find a definite place in the

prophylactic treatment of diphtheria, as numerous indications for the use of such a means occur at once to the clinician.

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II. THE DIPHTHERIA TOXIN SKIN REACTION

NATURE OF THE REACTION

In 1909 Schick found that if a solution of diphtheria toxin was applied to the skin in the same manner that tuberculin is used in the von Pirquet tuberculin test, it produced a reaction in many persons very similar to the tuberculin reaction. Later, by using the intracutaneous method, he was able to regulate the amount of toxin absorbed and this has led to a number of studies which are of both theoretical and practical importance.

The toxin used by Schick and his associates is a dilution of such strength that 0.1 c.c. equals 1/50 of the lethal dose for a 250 gm., guinea-pig. The lethal dose of the toxin which Schick uses is 0.005, and hence he injects 0.1 c.c. of a 1-1,000 dilution. In those who react an area of reddening and infiltration develops within twenty-four hours, reaching its maximum in forty-eight hours, and which heals with scaling and a characteristic central pigmentation. Although the reaction is similar to the local tuberculin reaction, its interpretation is directly opposite. The diphtheria toxin is a direct toxic agent and by control tests of the blood serum it has been found that *a negative reaction is always associated with the presence of diphtheria antitoxin in the blood* of the person tested. While

as a rule a positive skin reaction is an indication of the absence of antibodies, some persons react positively for some unexplained reason who possess a greater amount of antitoxin in the blood than 0.03 units per cubic centimeter.

It has been found that if a negative reaction follows the injection of a 0.1 c.c. of a 1-1,000 dilution of toxin, the individual tested has at least 0.031 units of antitoxin per c.c. in his blood when tested by Römer's method. A person with a higher concentration of antitoxin will react negatively to a smaller dilution of antitoxin and vice versa. Thus the outcome and the degree of reaction are dependent on two factors—the strength of the toxin used and the presence of antitoxin in the blood.

The specificity of the diphtheria toxin skin reaction is shown by its suppression or inhibition by the simultaneous injection of antitoxin in individuals who have previously given a positive reaction. In this connection the work of Schick and Maygar, who studied the simultaneous injection of diphtheria toxin and tuberculin, is interesting. Previous studies of tuberculin and toxin reactions in the same individual by Rolly, Entz and others, led them to conclude that the reaction to tuberculin was a general reaction toward toxic substances. Schick and Maygar tested 315 individuals with both tuberculin and diphtheria toxin at the same time and only obtained the same reaction (both positive or both negative) in 163 cases. In 153, or almost 50 per cent., the reactions were different.

THE REACTION AS A PRELIMINARY TEST FOR PROPHYLACTIC INJECTIONS OF ANTITOXIN

As noted above, it has been found that if a negative reaction follows the injection of 0.1 c.c. of a 1-1,000 dilution of toxin, the blood serum contains at least 0.031 units of antitoxin per cubic centimeter. This is sufficient to afford immunity against infection with the diphtheria bacillus under ordinary circumstances.

A large number of tests on normal individuals have been made and the reactions obtained are given in the following tabulation. Many of these have been controlled by Römer's method.

	Total	Positive	Negative	Per Cent. Negative
New-born infants.....	291	10	275	93
First year of life.....	42	18	24	57
2 to 5 years.....	150	95	55	37
5 to 15 years.....	264	131	133	50

It is thus seen that a large percentage of individuals are immune to diphtheria. The age period giving the lowest percentage of negative reaction (2 to 5 years) is the same as that in which the highest incidence of clinical diphtheria is found. As far as could be deter-

mined the reaction remains the same for a period of at least four weeks.

The practical value and application of such results is obvious. Whenever it is necessary to immunize against diphtheria for prophylactic purposes, a diphtheria toxin skin test will show in twenty-four hours those who already possess immunity. Only those reacting positively to the toxin require passive immunization with antitoxin. As roughly 50 per cent. possess immunity this will reduce the number of prophylactic injections one-half. The real extent of such a reduction is seen when one considers that, according to Park, two injections for immunization are given in New York City to one for curative purposes. The liability to sensitization to horse serum is coincidently lessened to the same extent.

AS A TEST FOR ANTITOXIN CONTENT OF THE BLOOD

In the past the manner of measuring the quantity of diphtheria antitoxin in the blood has been indirect. The blood serum of the individual tested has been added in different dilutions to a definite quantity of toxin and injected into guinea-pigs. The blood content in antitoxin is calculated from the dilution which just neutralizes the toxin.

Schick and Michiels having once found that 0.1 c.c. of a 1-1,000 dilution of their toxin requires 0.031 units per cubic centimeter of toxin in the blood to suppress the toxin reaction, have been able to test directly the quantity of antitoxin by varying the strength of the dilution. If a negative reaction follows a 1-1,000 dilution, a 1-500, 1-200, etc., dilution was used until a strength was reached at which a positive reaction occurred. It is necessary first to find the strength of a toxin on guinea-pigs, after which it may be used directly in different dilutions.

USE IN ANTITOXIN EXPERIMENTS

Until the elaboration of the toxin skin reactions, all experimental work in regard to the relations of toxin to antitoxin have been carried out on animals, and our ideas regarding the administration and efficacy of antitoxin in man have very largely been based on animal experimentation. Schick and his associates have carried out a large number of experiments on children in regard to these questions.

Children who reacted positively to a toxin skin test were given antitoxin in different amounts and at the same time a toxin reaction was made. Fairly large quantities of antitoxin were needed to suppress the reaction (2,000 units intramuscularly). In another series of experiments toxin tests were made at two and three hour and

longer intervals and the effect of large doses of antitoxin noted. Practically no effect could be made on toxin injected more than three hours before the administration of antitoxin. Reactions from injections of toxin made forty-eight, seventy-two and ninety-six hours after the administration of fairly large quantities of antitoxin were suppressed.

Schick comes to the conclusion that the action of antitoxin is chiefly immunizing, and that injections should be made as early as possible and either intravenously or intramuscularly. The value of antitoxin in severe cases depends on whether a lethal dose of toxin has been absorbed before the antitoxin was administered.

OTHER USES

A few attempts have been made to produce active immunity by repeated injections of small amounts of toxin. Three injections of 0.1 c.c. of a 1-500 dilution were made two weeks apart. As no weakening of the reaction was observed at the third injection the result was looked on as unsuccessful. No indications of hypersensitiveness were observed.

As there is no antitoxin present free in the blood in acute diphtheria, the use of the reaction for diagnostic purposes has been suggested. Thus in a suspected case or questionable diagnosis a negative reaction — indicating the presence of antitoxin — would speak against the diagnosis of diphtheria.

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1806 Locust Street.

DIGESTIVE DISTURBANCES OF ARTIFICIALLY FED BABIES *

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During the last twenty years there has been much change in the interpretation of symptoms occurring in the digestive diseases of babies. The older physicians regarded acute and chronic gastro-intestinal disorders as being always due to diseases of the stomach and intestines. Fermentation dyspepsia, fermentation catarrh, gastro-enteritis, cholera infantum, dysentery, ileocolitis. These were compared with similar conditions in adults, allowance being made for the slighter resistance in babies which favored a severe course of the disease.

Widerhofer made a classification on anatomic grounds, dividing the various symptom-complexes into:

1. Purely functional.
2. Catarrhal.
3. Ulcerative-inflammatory lesions of the intestines.

These were clinically distinguished by the character of the stools and by the constitutional symptoms; thus, he speaks of acute and chronic dyspepsia, enterocolitis and enteritis with inflammatory lesions in the large intestine. A fourth form was the murderous cholera infantum.

This description is most valuable. Yet there were many intermediate and transitional forms and but few clear-cut typical cases.

Although severe inflammatory infections of the intestine do exist, yet they are less frequent in importance and frequency than purely functional disorders. It is also true that persistent functional disturbance may cause anatomic changes.

In all varieties of cases a uniform and normal condition of the intestines may exist where the old teaching would suggest severe organic lesions. With some digestive disorders the symptoms and course are more dependent on general visceral changes than intestinal inflammation.

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* Submitted for publication, June 13, 1914.

* This article is a condensed translation of Finkelstein and Meyers' treatise on diseases of the digestive tract in Feer's new book on Diseases of Children.

Thus, bacterial infection does not play the important rôle formerly supposed. Many symptoms are due more to alimentary food-intoxication than to bacterial toxins. The vomiting, fever, diarrhea are usually due to disturbed metabolism; hence the term introduced by Czerny "Disturbance of Nutrition."

Formerly great importance was ascribed the bacteria and bacterial products in polluted milk, yet this factor is of really slight importance compared with improper dosage or character of food. The maintenance of health and development is intimately associated with good functional energy and normal tolerance. A child with good tolerance may be injured by under-nourishment or over-nourishment; with correct feeding a baby may become ill through primary weakness of digestion, a misproportion between the demands of appetite and the process of assimilation.

Inanition also plays a serious rôle in infantile pathology. The food may be too highly diluted or lack important food element, as in gruels and proprietary foods. There are also disturbances due to over-nourishment; the digestive functions are overworked and weakened. The tolerance has been exceeded even if the quantity and character of the food recipe be theoretically correct. Overfeeding may cause sickness in a feeble baby. The diet may not exceed the physiologic need, yet it may make great demands on an especial function, leading to its exhaustion.

A breast-fed baby with careless handling thrives better than a bottle baby with the most correct regimen.

GENERAL PATHOGENESIS

Most children are easily fed. A few show uncertain digestion and have a stormy infancy. Overfeeding leads to delayed assimilation, food-stagnation and bacterial decomposition. Sickness from relative underfeeding is seen in weakly children of the neuropathic exudative diathesis. With artificially-fed babies tolerance is easily exhausted, and cow's milk mixture with the exact chemical formula of human milk has a radically different effect and often injuriously affects a baby. The difficult digestion of casein is a myth, shown by the analysis of stools, but there is no doubt of the occasional injuries by fats and sugars. This reaction is not primary, but is secondary to fermentation of the whey. The whey of human milk, on the contrary, acts as a stimulant to digestion. The fat and albumen of cow's milk in whey will cause sickness, while if the same fat and albumen be mixed in human whey, the child will thrive.

The poor digestion of fats and sugars is due to pathologic fermentative processes, resulting in acid formation causing a local reaction on the intestinal mucosa, increased peristalsis and secretion of mucus. The child's health deteriorates and the protective processes of metabolism are lessened and poisonous substances formed.

Bacterial poison causes cell injury of the internal organs as well as of the intestinal tube.

Excessive atmospheric heat hinders heat evaporation and injures the working capacity of the cells; symptoms like sunstroke often appear, coma and hyperpyrexia, which latter is relieved by hydrotherapy. Artificially fed children do not resist heat well.

Contaminated milk was formerly held to be the most important and frequent cause of diarrheal disease, but intestinal fermentation is usually endogenous and will develop in a sterile food from primary digestive incapacity; the present theory is that within certain limits heat has a damaging effect on the child, reducing digestive tolerance so that food injuries easily occur. A bottle baby with disturbed nutrition has less heat resistance than a child which is nursed.

GENERAL SYMPTOMATOLOGY

More variety exists than was formerly supposed. The symptoms affect not only assimilation and tissue-building, but also internal metabolism and heat production and regulation, therefore the description should not be limited to vomiting and diarrhea. We observe albuminuria, cerebrospinal irritation, anomalies of heart and lungs, which often may be relieved by a change in diet.

All of us admire the fine color and beautiful contour of a healthy baby. It has normal stools and by the continual formation of antibodies effectively resists infections, which, if they do occur, run a benign course. It also has a breadth of tolerance for food, thriving on all sorts of mixtures and passes easily through the extreme heat of summer.

A baby with disturbed digestion shows emaciation, increased or diminished muscular tonicity, pallor, excitability or apathy, fluctuations in temperature and suffers a lowering of resistance against bacterial influence. Alimentary fever is often confused with infection; it may be distinguished by its course and its disappearance by a change in food.

Children with disturbed nutrition show a lessened tolerance for food. A healthy child with an increase in diet will react by a gain in weight. A sick child will show a paradoxical reaction. An increase of food aggravates its symptoms and causes a loss in weight.

Example.—Three children (1) healthy; (2) slightly disturbed; (3) severely disturbed. Thirty grams cane sugar added to twenty-

four-hour quantity of food for two or three days. Healthy child increased in weight. Second child slightly ill with mild fever, diarrhea and a stationary weight. Third child has a temperature of 100.5 F., violent diarrhea and lost several hundred grams.

This reaction to food is the measure of the reduced functional power. The more the patient is disturbed by small increases of food, the more serious is its condition. Such children are very sensitive to infections, heat, etc.

CLASSIFICATION: NUTRITIONAL DISTURBANCES

Czerny:
- Disturbances: Alimentation; various foods; injuries.
- Disturbances: Infection; polluted milk.
- Disturbances: Congenital weakness of constitution.

Finkelstein:
- Mild forms without destructive processes
  - (1) balance disturbance
  - (2) dyspepsia
- Severe destructive processes
  - (3) decomposition
  - (4) intoxication

Also, disturbances from inanition — feeding on gruels, proprietary foods; treatment by repeated periods of starvation.

Infection, heat, etc.

Whole question is of lessening of digestive tolerance.

Balance disturbance — lessened tolerance for fat in milk.

Symptoms: Insufficient gain in weight; firm stools.

Dyspepsia
- Lowering of tolerance for fat of milk
  - Symptoms: Insufficient gain in weight; vomiting; diarrhea; sometimes constipation.
- Lowered tolerance for carbohydrates
  - Vomiting; diarrhea; colic; paradoxical reaction for food.

Decomposition, Marasmus, Atrophy
- Severe damage for digestion of fats and carbohydrates
  - Abrupt loss in weight; subnormal temperature. Slow pulse; paradoxical reaction; strong reaction to heat and infection.

Intoxication
- A condition developing on the basis of dyspepsia and marasmus if large quantity of food containing whey and sugar is given.
  - Fever; cerebral disturbance; albuminuria; glycosuria, deep breathing, vomiting and diarrhea.

BALANCE DISTURBANCE — CZERNY — SLIGHT MILK INJURY

Retarded development without marked symptoms of illness. The weight curve rises slowly, fluctuates and remains stationary or diminishes. The patient appears smaller than the normal child; has a poor color, flabby muscles, distended abdomen, irritable disposition; the stools are dry and gray (soap stools); it occasionally vomits; is easily infected.

Two types exist. 1. Fed chiefly milk.
2. Milk and carbohydrates.

The child may always show poor progress on milk mixture or the food may contain too much milk and too little carbohydrate.

The baby may do well for a while or may have a congenitally weak tolerance, or one reduced by an infectious illness.

Pathogenesis.—Cream is usually the injurious food element, and there is often rapid improvement on a diet poor in fat and rich in carbohydrate, which improvement is arrested by adding fat again. It is probable that the low digestive power for fats is secondary to weakening of intestinal function, through abnormal fermentation of sugar of milk; a rapid cure may often be effected by substituting another carbohydrate, dextri-maltose or a cereal gruel.

Sometimes an injured tolerance for carbohydrates causes the condition to advance to dyspepsia.

There is a change in metabolism, chiefly in fat absorption, the alkaline secretions of the intestine are increased and combined with fatty acids; there is increased secretion of mucus; the stools are of a whitish color; the bilirubin is changed to urobilin. To produce a soap stool the large intestine must be strongly alkaline — soap stools are not pathologic and may be observed with normal development.

Diagnosis.—If a baby with sufficient nourishment, 100 calories per kilo of weight per day, with no diarrhea or vomiting makes no progress, one may make a diagnosis of disturbed balance. The condition may be confused in children reduced in weight by illness, for convalescents have a greater need of nourishment, often taking 120 to 140 calories per kilo per day. One must also consider mild cases of decomposition in whom a change in food has stopped a diarrhea. These cases show a paradoxic reaction, dropping in weight if food in increased.

Lastly, there are anomalies of growth from unknown constitutional causes which defy the most skillful feeding.

Prognosis.—With appropriate dietetics the prognosis is very good. The number and quantity of meals should be regulated, the milk reduced and the carbohydrates increased. The type due to excess of milk is most easily treated by adding a little cereal gruel, a malt food or two carbohydrates in proportion of 2 to 5 per cent. of the total recipe. If one carbohydrate fails, try another; not more than five meals daily; quantity of food should be watched until the tolerance is known. Malt soup is often effective in obstinate cases.

Recipe for Malt Soup.—50.0 wheat flour, ⅓ liter milk, malt extract 100.0, ⅔ liter water. Mixed, cooked, strained.

Or buttermilk, clean and not too acid, mixed, with a flour-gruel and cane-sugar are excellent for younger babies.

Dextrimaltose may be considered constipating. From 2 to 3 per cent. of carbohydrates should be given. With proper dietetics, increased weight and good stools should soon occur. If malt-soup or buttermilk is given, after six or eight weeks an effort should be made to place the baby on a normal diet. A cure is established only when a suitable milk mixture agrees with the baby.

If the baby's condition still remains unsatisfactory in spite of carbohydrates, the outlook is less favorable; low carbohydrate toleration leads to fermentation. If all means of artificial feeding fail, breast milk is advised as in the treatment of dyspepsia.

DYSPEPSIA

Dyspepsia may be primary or a result of primary balance disturbance. The digestive capacity of the stomach is insufficient for the food given. There results pathologic fermentation, producing increased peristalsis and diarrhea — the chief symptoms of dyspepsia. The baby suffers from poor appetite, vomiting, slight motor insufficiency of the stomach, the free HCl is diminished, the volatile fatty acids increased, the abdomen is distended and there is colic. The stools are increased in number, are thin, contain mucus; the odor is foul and sometimes sour. The color of the stools is green; the bilirubin is oxydized to biliverdin. As a result there occurs the following: Fat soap stools; small white or yellow lumps, on warming, producing fat crystals; gruel (flour) stools are pasty and foamy; blue with iodin; red with eryfrodextrin. As to the question of undigested casein stool, little yellow lumps formerly supposed to be casein, are really fatty acids and bacteria.

Only after feeding raw milk will stiff cylinders of casein appear in the stools.

Formerly dyspepsia was divided into fat and starch (*Mehl*) dyspepsia. This classification is not justified for the appearance of undigested food elements in the stool does not prove that this element is the cause of the trouble. Thus, fat diarrhea does not come from primary insufficiency for fats, but from carbohydrate fermentation, causing increased peristalsis and fat in the stools. By withdrawing carbohyrates the fats can again be utilized.

Pathogenesis.—The local symptoms of dyspepsia are due to the increased production of fermentative acids from pathologic decomposition of carbohydrates, sugar and starch. Again, it may be emphasized that casein, so far from injuring the intestines, is most effective in combating an acid fermentation and exercises a curative influence.

The carbohydrates used in feeding have important differences as to fermentation. The sugar of milk ferments most easily; cane sugar

less so; dextrimaltose is slow to ferment, hence the apparent constipation following its use.

The tolerance of an intestine for carbohydrate as well as fats depends much on the fluid in which they are dissolved or suspended. The same quantity of sugar in undiluted whey will more quickly cause dyspepsia than the same amount in water or in diluted whey. As a cause of dyspepsia in bottle babies, whey, even if regarded as free from sugar, plays an important rôle.

We have an acute dyspepsia and chronic dyspepsia, a temporary or persistent lowering of digestive function.

Diagnosis.—The chief thing in diagnosis is to separate dyspepsia from mild enteral and paraenteral infections which may also cause an irritation of the intestines. It must be remembered that infections are often associated with disturbed nutrition. An infection is present when temperature and mucous stools continue in spite of reduction in carbohydrates.

The physician is cautioned not to regard intestinal irritation from an infection as being due to fermentation, and institute a long, unnecessary course of underfeeding.

A dyspepsia being diagnosed, the physician must consider whether he is dealing with a previously healthy child or with a marasmus patient. This is most important as to treatment and prognosis. The history should be reviewed; the first attack of diarrhea is probably dyspeptic; recurring diarrhea and loss of weight indicate that the digestive strength of the child has suffered, and point to marasmus.

Prognosis.—In a previously healthy child the prognosis is good; recurring attacks in very young babies, or in emaciated patients are most serious.

DIETETIC TREATMENT

The safest treatment in all forms of dyspepsia is human milk; it is most necessary to babies in the first weeks of life where artificial feeding is so dangerous. The dose of woman's milk should be generous, although at first caution is advisable.

In acute dyspepsia, in a fairly healthy patient where the cause of attack is a misproportion between the child's appetite and his digestive power, there should be a short period of fasting, with copious draughts of saccharin tea for six or twelve hours; only exceptionally for a longer period. Complete emptying of stomach and intestine is effected by lavage and irrigation, and laxatives. One-third of the usual amount of food, well diluted, should be given. The nourishment should be rapidly increased so that the period of underfeeding may be as short as possible.

As to the kind of food, the offending food elements, fats and carbohydrates, should be avoided; also the injurious effects of whey should be remembered. A thin gruel, mixed with skim milk, is advised, and later dextrimaltose may be added. No sugar of milk. Skim-milk and buttermilk are usually well digested.

This dietetic treatment causes a sudden fall in weight, later a stationary condition, normal temperature and fewer stools.

After a few days the child's tolerance for carbohydrates should be tested.

Many patients, however, do not respond to this treatment, and the diarrhea continues. These children either have an infection or are on the verge of marasmus. To continually underfeed these children is to kill them.

Finkelstein recommends the use of albumen milk for these cases.

Children with chronic dyspepsia do badly under continued underfeeding. There is no acute depression, but a chronic weakness of tolerance and the continual trauma of hunger is injurious. The best carbohydrate is 2 to 3 per cent. dextrimaltose. If no improvement in the stools occurs, use human milk or albumen milk.

DECOMPOSITION — ATROPHY — MARASMUS

The chief symptom is the loss in weight which sinks slowly, at times very suddenly. The baby is emaciated, has a senile appearance, the abdomen is distended, the complexion gray.

At first the child is excitable, crying and taking food greedily; later it is languid, apathetic. The inclination is very noticeable to slow, irregular heart action and subnormal temperature, which may be later associated with edema and cyanosis.

The urine contains no albumen or sugar. The stools are dyspeptic and frequent; sometimes fluid, sometimes solid. The firm stool indicates an arrest of the process. Often the feces contain a large amount of fat. Occasionally the feces are dark red or black from hemorrhage from a peptic duodenal ulcer.

Such patients are most sensitive, showing an immediate severe reaction to unsuitable food, infections, etc. A cold, bronchitis or hot weather may cause high fever and collapse; purulent infections of the skin, pyelitis and pneumonia indicate the diminished immunity of the child.

Decomposition in very young babies, if not quickly improved by diet, is soon fatal. With older children there are periods of remission and improvement. A not unusual ending is a fatal hemorrhage from a duodenal ulcer. Some children die with symptoms of alimentary intoxication. Death may be quite sudden, especially after a therapeutic

hunger period. A hopeful case may succumb quickly to an intercurrent infection.

Pathology, Metabolism.—It was formerly supposed that marasmus was an inanition due to destruction of the digestive glands from chronic inflammation, but the intestine of an atrophic child may show normal anatomic relations. Decomposition is probably due to extreme functional depression as shown by the paradoxic reaction in feeding. The more nourishment the baby swallows the more rapidly it emaciates. A sudden drop of a pound or two is due to loss of water and salt. The melting of tissues goes on more slowly.

The prognosis depends wholly on the dietetic treatment. If errors are avoided and the case is not seen too late, very bad cases may be cured; if one-third of the original weight is lost, the condition is hopeless.

Diagnosis.—We must distinguish marasmus from the symptomatic emaciation of tuberculosis and other cachexias and also from inanition. Mild forms of decomposition may be confused with balance disturbance or dyspepsia. The history of recurring diarrhea or rapid loss in weight is important. The final test is the behavior of the digestion in feeding. With a fairly strong food mixture a marasmus child will show fever, diarrhea and a drop in weight.

Treatment.—The cure of the decomposition is possible if the fermentation which causes the deterioration in digestion is removed and the tissues are supplied with water and salts. Often the physician commences treatment as he does in dyspepsia, with a hunger period and small and increasing doses of food, but a marasmus child cannot endure starvation and should, if possible, be nursed. The danger of hunger is exhaustively considered under "Inanition." If insufficient food is given, the digestive strength is ruined.

With human milk the subtle curative agent is in the whey, which is somewhat offset by the difficult digestion of fat and sugar. If one gives too much the baby is upset; if too little, it grows rapidly worse. Finkelstein gives the breast milk in a bottle, 200 to 300 grams daily in eight to ten feedings, with copious draughts of saccharin tea, without a hunger period, to avoid the fatigue of nursing. The milk is continually increased so that after a week the baby is taking from 100 to 130 or 150 calories per kilo of weight daily.

At first the child may grow paler and thinner, but not after the second week. Then follows a period when the general condition improves, but there is no gain in weight. No change in the wet-nurse should be made. We are dealing with a period of repair. Cells are constructed slowly with human milk, which is poor in albumen and salts.

In a month the improvement may be hastened by one meal daily of malt soup or of buttermilk, with a small amount of dextrimaltose.

A complete restoration to health will not occur earlier than two or three months; then ordinary food mixtures may be tried. It must be remembered that the child may have an idiosyncrasy against cow's milk.

If no human milk is available, mixtures of skim-milk or buttermilk diluted with a thin gruel may be used; often an increase in strength of the food will occasion a serious diarrhea. Finkelstein strongly recommends albumen milk in such cases.

The idea of albumen milk is to limit the dangerous acid fermentation by lessening the percentage of sugar of milk in the whey.

3.0 Albumen sweetened with saccharin. Maltose should be added in the proportion of not less than 3 per cent.
2.5 Fat; 450 calories per liter.
0.5 Sugar.

A great advantage of albumen milk is that sufficient food may be quickly given without danger of fermentation. The risk of underfeeding and inanition is thus avoided. The amount of albumen milk may be quickly increased to 180 or 200 gm. per kilo daily. Total daily quantity should not exceed a liter. After a day or two the stools are firm and fewer in number. The weight curve ceases to sink, becomes stationary and then increases.

With advanced marasmus, to evacuate the intestines quickly a six- to twelve-hour hunger period with tea may be advisable, followed by 200 to 300 grams a day of albumin milk in ten doses; the amount is quickly increased and the number of bottles diminished. If the fall in the weight curve is not arrested in eight days, dextrimaltose should be increased.

If this technic is followed, the number of failures is surprisingly small.

Lack of success is due to:

1. Too slow an increase, producing inanition.
2. Too late and insufficient carbohydrate.
3. Omitting carbohydrates if the stools do not improve.
4. Reducing or withdrawing carbohydrate if a recurrence of diarrhea, fever or of emaciation occur.

In only extreme conditions should these changes be made and only for a short time.

Feeding with albumen milk should continue in young babies ten weeks; older babies six to eight weeks. After this, ordinary food mixture may be digested. A cure may be considered complete when after a course of albumin milk, followed by a milk mixture, the child continues to thrive.

INTOXICATION

An alimentary intoxication is a general toxic condition in a child with disturbed digestion who is given food exceeding its digestive power. The lower its functional tolerance is, the smaller the amount of food is which will produce the intoxication.

Symptoms.—The first symptom is fever, whose alimentary nature is proven by the fact that the temperature quickly becomes normal if nourishment is withheld. With the mildest type there is a slight rise in temperature; with a severe attack the fever may be 104, 105, 106 F.

The alimentary fever may be the only symptom or may be associated with diarrhea; usually the patient loses weight, is languid and shows signs of kidney irritation. These early symptoms are immediately, or after one, two or three days, followed by extreme depression diarrhea, nervous symptoms, deep breathing, albumin and casts in the urine, and a serious drop in weight. At first the nervous condition is one of languor and somnolence. The child, if aroused, sinks immediately back into its lethargy. The face twitches, the eyes are sunken. In the worst cases the patient lies in a coma, or has general convulsions, transitory paralysis. Usually there is high fever, but a bad case of marasmus may show no feverish reaction.

The respiration is of a toxic type — deep, rapid, like a hunted animal. The stools are frequent; at first contain food débris, later are watery, greenish-yellow; are mixed with mucus. They become strongly alkaline from the abundant alkaline intestinal secretion.

Vomiting is often a serious symptom, recalling the clinical picture of the old-fashioned cholera infantum. The weight may sink from 500 to 1,000 grams in a few days. The skin is dry, shrunken, features drawn, fontanelles sunken; the muscles may be contracted and cramped.

The rapid loss of fluids quickly leads to collapse. The pulse is small, the heart weak, the extremities cold and cyanosed; the urine contains sugar from an alimentary glycosuria.

There may be a leukocytosis, reaching 30,000.

The great variety of symptoms show how many organs are involved. We may separate several different symptom-complexes — a comatose cholera form and cerebral type.

Pathologic anatomy.—The morbid changes are not extensive and do not correspond with the serious illness. In the stomach and intestines there is a serous or serohemorrhagic catarrh. Peyer's patches are swollen and hyperemic. Microscopically the wall of the gut shows a round cell infiltration.

Etiology.—The great similarity of an infantile alimentary intoxication to true cholera and cholera nostras of adults caused the older

physician to speak of cholera infantum and to ascribe the malady to an infectious origin, accusing contaminated milk. While such symptoms may occur in the course of parenteral and other infectious diseases, yet no pathogenic specific germ has been found to explain the symptom-complex. On the contrary, the most careful clinical observation shows that the condition is a food intoxication from exceeding the subject's digestive tolerance, and the origin of the disease is due to a decomposition of food. The use of polluted milk is therefore not essential, since the intoxication may originate from aseptic food.

The fact that food is the cause of the malady is proven by the effect of withdrawing food and giving water freely.

In all cases not complicated by an infection, the temperature at once falls; if food be taken again the fever returns. The fever-producing agents are the carbohydrates combined with whey constituents. If these substances are given in large quantities the fever is complicated by a toxemia. Large amounts of fat have also a toxic effect. This fat intoxication is, however, not primary, but is due to a sugar injury, which has severely damaged the function of metabolism.

Nature or Toxic Condition.—An intoxication is a result of an insufficient intermediate metabolism associated with an acidosis. The damage to carbohydrate metabolism shows itself by an alimentary glycosuria; this disturbance is increased by hunger and loss of alkalies by a diarrhea.

Formerly it was asserted that intestinal stagnation produced a toxic agent of bacterial origin, but experimental observations show that food crystaloids cause fever and poisoning by physical osmosis. The thickening of blood is also of great importance.

Diagnosis.—Alimentary fever is distinguished from infectious fever by a therapeutic test, withdrawing food or by reduction of the whey and sugar in the mixture.

In a food intoxication the temperature falls by crisis unless the system is fatally damaged. In marasmus the patient may die of hunger with no sign of detoxication. Pure infections or infectious complications are not freed from fever by hunger metabolism.

Finally, with a very well-nourished child the detoxication may be slow and incomplete. (No explanation.)

Prognosis.—The outlook depends less on the severity of the symptoms than on the duration of the toxemia and on the previous condition of the child.

Prompt and correct treatment will cure most patients quickly. Thus, a baby who sickens on sweetened buttermilk may have threatening symptoms and very rapidly improve. A long period of intoxication is naturally exhausting. A marasmus baby will often die without relief.

Treatment.—With bad toxic symptoms the complete withdrawal of food cannot be avoided. Fluids should be freely supplied in the form of saccharin tea. To relieve the loss of fluid, salt solution is recommended. It has the disadvantage of sometimes causing fever. (Denied.)

Lavage of the stomach and intestine is useful, but not indispensable.

Laxatives should not be used in diarrheal cases.

As stimulants, camphor, caffein, digitalin, adrenalin, cognac, are well spoken of. High temperatures are to be combated by hydrotherapy. With a cold skin, hot mustard baths are effective.

With favorable, uncomplicated cases the starvation will, in twenty-four to thirty-six hours, cause a complete disappearance of the intoxication. The stools are less frequent; the baby is thin but placid. Feeding must now be commenced, at first in small but increasing amounts. Fluids must literally be pushed into the patient.

Human milk is the best food. The earlier the child is rescued from inanition the better; on the other hand, over-feeding will quickly produce a relapse.

Human milk may at first be given in a bottle; later the baby may be nursed.

If artificial food be used, the injurious fats and sugars should be avoided. Skim-milk, buttermilk are useful. The first doses should be 5 gm., five to ten times a day. The dietetic treatment is similar to that in dyspepsia.

Albumin milk is much used by Finkelstein. It has no tendency to ferment and can be digested in large quantities.

An emphatic warning is given, that mixed forms of intoxication and marasmus babies do not endure starvation, and after a few hours on tea they should be fed either human milk or albumin milk.

NUTRITIONAL DISTURBANCES IN BABIES FROM LACK OF FOOD

We see frequently the clinical picture of inanition and its consequences in children who have been given insufficient food on account of primary intestinal disturbances. Healthy babies or those who are only slightly ill respond to a considerable reduction in food by constipation and a loss in weight. If the underfeeding is continued the patient will have subnormal temperature and slow pulse.

The similarity of this condition with decomposition is unmistakable. At first, hunger inanition is distinguished from decomposition in that an increase in food brings up the weight quite rapidly, but finally the organism is so weakened that digestive tolerance is much reduced. This is observed in the convalescence from severe spastic pyloric stenosis after vomiting has ceased.

If the patient is given large quantities of food, even of human milk, it may lose in weight — a severe paradoxical reaction. A child dying of pyloric spasm sinks into a terminal toxic coma as in marasmus.

More frequently one sees bad consequences of inanition in severe disturbance of nutrition. Here food has been diminished or withdrawn far too long a time and without knowledge of the great danger.

With such children hunger and underfeeding has a terrific effect — a drop in weight, slow pulse, subnormal temperature, collapse and even death.

There is often a permanent weakening of digestion, so that the amount of food that is assimilated before the hunger period will now cause serious symptoms. In a severe case a short period of starvation gives the patient a strong push downward. A baby grows steadily weaker with moderate underfeeding, while repeated hunger periods result in a hopeless condition.

It may, therefore, be laid down as a fundamental principle in the prophylaxis and therapeutics of disturbances of nutrition that starvation and underfeeding can indeed remove toxic symptoms and fermentation of alimentary origin, but they may also cause far-reaching damage, and that this starvation treatment must be reduced to the minimum of necessity.

QUALITATIVE INANITION

This is a form of malnutrition in which the nourishment lacks important food elements and has an insufficient caloric value; as a result, the vitality is seriously damaged and poor tissue is formed.

The most common type of inanition occurs in babies fed largely or wholly on starchy gruels. The gruel has been prescribed to relieve diarrhea and has been continued by the mother. For a while the body shows no signs of illness, and may gain in weight from the retention of water in the tissues by the carbohydrate diet. The flesh is soft and pasty and the patient may have a muscular hypertonia — latent tetany.

1. The atrophic type is seen where starch gruel has been given without salt. It is distinguished from starvation inanition by the dryness of the tissue and the hypertonicity of the muscles.

2. The hydremic form is observed where the gruel has been salted and the weight is increased by water retention. The face is pale and swollen, the flesh is flabby and often edematous, without kidney irritation. Occasionally great muscular rigidity may be seen.

The stools vary with the carbohydrates — are foamy, mucous, liquid or solid; sometimes the patient has a colitis.

Wide fluctuations in the weight curve are characteristic.

If the child has a slight infection the weight may drop from several grams to a kilo. Purulent skin infection, inflammation of the cornea, conjunctiva, pneumonia or pyelitis may show the lessened resistance to bacterial infection.

The disturbances from the exclusive use of starch gruels result from the lack of essential tissue-building food elements — fats, albumin, salts. A serious consequence is the lowered production of antibodies and reduced immunity.

Prognosis.—The younger the baby and the longer the incorrect feeding has been carried out, the worse is the outlook.

Prophylaxis.—Qualitative inanition is prevented by a sensible diet, when starchy diet is ordered for therapeutic reasons to relieve convulsive symptoms or stop a diarrhea. The danger of starch inanition should be remembered with spasmophilia. Albumin preparations, fats and salts may be used. With a diarrhea, gruels should be used for only a short time without adding milk.

Treatment.—With very young babies human milk is best at first in minute doses. In general, whole milk should be added to gruel. Finkelstein advises albumin milk. The gruel should be gradually reduced, lest a terrific drop in weight occur.

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